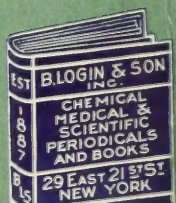

EST
1887
B. LOGIN & SON
INC.
CHEMICAL
MEDICAL &
SCIENTIFIC
PERIODICALS
AND BOOKS
29 EAST 21ST ST
NEW YORK

TRANSACTIONS

of the

American Ophthalmological Society

Fifty-First Annual Meeting

NEW LONDON, CONN., 1915

VOL. XIV, PART I

332538
27. 10. 36.

Philadelphia
American Ophthalmological Society
1915

2 250 ampl

RE
1
A55
V.14

PRESS OF WM. F. FELL CO.
PHILADELPHIA

TABLE OF CONTENTS.

PAGE

List of Officers and Members v

Necrology 4

Minutes of the Proceedings 21

A Review of the Glaucoma Question, with Special Reference to Newer Conclusions of Its Etiology and Departures of Its Surgical Treatment: Dr. Robert Sattler 38

A Report of Several Cases of Chronic Simple Glaucoma Treated by Iridotasis: Dr. David Harrower 51

Tenotomy of the Inferior Oblique Muscle: Dr. Wm. Campbell Posey ... 63

Rosacea Keratitis and Certain Other Forms of Marginal Keratitis, Neuropathic in Origin. Treatment by Pericorneal Neurotomy: Dr. F. H. Verhoeff 89

Some Experiences in Enucleation under Local Anesthesia: Dr. William M. Sweet 98

Massive Granuloma of the Sclera (Brawny Scleritis), with the Report of an Unusual Case: Dr. George S. Derby. Pathologic Examination by Dr. F. H. Verhoeff 111

Salvarsan in Interstitial Keratitis and in Optic Nerve Lesions: Dr. Arnold Knapp 124

The Ives-Langdon Photometer: Dr. H. Maxwell Langdon 144

A Review of the History of the Electric Ophthalmoscope, with a Demonstration of a New Ophthalmoscope, which Consists of a Rearrangement of the Parts of Antecedent Instruments: Dr. J. Herbert Claiborne 150

A Simple Modification of the Berger Loupe: Dr. Edward A. Shumway .. 158

Nagel's Anomaloscope for Testing Color Vision: Dr. Charles H. Williams 161

The May Electric Ophthalmoscope: Dr. Chas. H. May 165

Stronger Eye Magnets: Dr. Walter B. Lancaster 168

A More Exact Method of Measuring Fields of Vision and Arcs of Rotation: Dr. Lucien Howe 183

Ocular Phenomena Accompanying Three Cases of Gastro-Intestinal Disorder: Dr. Hiram Woods 189

Lumbar Puncture and Examination of the Spinal Fluid in Affections of the Eye: Dr. James B. Ayer (by invitation) 205

Tuberculous Meningitis with Special Reference to the Tubercle of the Choroid and Its Pathologic Manifestations: Dr. Frederick Tooke .. 218

PAGE

Malignant Uveitis Treated with Thyroid Extract: Dr. James Bordley, Jr. 232

Progressive Essential Atrophy of All Layers of the Iris: Dr. G. E. de Schweinitz 250

The Ophthalmias of Palestine: Dr. Harry Friedenwald 264

Pemphigus of the Conjunctiva.—Report of Two Cases: Dr. Edward Stieren 302

Report of a Case of Colloid Degeneration of the Upper Lid: Dr. Arthur J. Bedell 313

A Case of Epibulbar Sarcoma: Dr. Howard F. Hansell 320

Multiple Melanotic Nevi of Conjunctiva, with Malignant Invasion of the Corneal Limbus: Dr. F. P. Capron 322

Report of Two Cases of Metastatic Carcinoma of the Choroid and One Case of Myxosarcoma of the Orbit: Dr. John E. Weeks 326

A Case of Primary Intradural Tumor of the Optic Nerve: Dr. Charles R. Heed 331

A Primary Tumor of the Optic Nerve Successfully Removed, with Preservation of the Eyeball, by the Krönlein Method: Dr. Arnold Knapp 335

A Contribution to the Subject of Tumors of the Eyelid and Orbit: Dr. G. E. de Schweinitz 341

Hereditary Posterior Polar Cataract, with Report of a Pedigree: Dr. S. Lewis Ziegler and Dr. J. Milton Griscom 356

Family Degeneration of the Macula Lutea, with a Suggestion as to Its Cause: Dr. Brown Pusey 364

Retinal Disease with Massive Exudation and an Allied Condition: Report of Two Cases: Dr. William Zentmayer 370

Spontaneous Reattachment of the Retina after Twenty-two Years' Duration; Report of Case with Remarks: Dr. A. E. Davis 376

Anomalies of the Accommodation Clinically Considered: Dr. Alexander Duane 386

Nervous Symptoms from Contusions of the Eye: Dr. Edward R. Williams 402

American Ophthalmological Society.

FOUNDED 1864.

OFFICERS.

1915–1916.

President,	Dr. G. E. de Schweinitz,	Philadelphia, Pa.
Vice-President,	Dr. Peter A. Callan,	New York, N. Y.
Secretary-Treasurer,	Dr. Wm. M. Sweet,	Philadelphia, Pa.

MEMBERS.

* Associate Members. O. M. Original Members.

Elected.	Name.	Residence.	Place.
1896	Dr. A. E. Adams,	Balmville, Cor. North,	Newburg, N. Y.
1915	*Dr. Ellice M. Alger,	40 East 41st Street,	New York, N. Y.
1899	Dr. A. N. Alling,	257 Church Street,	New Haven, Conn.
1896	Dr. G. W. Allyn,	Empire Building,	Pittsburgh, Pa.
1882	Dr. A. Alt,	Metropolitan Bldg.,	St. Louis, Mo.
1883	Dr. J. A. Andrews,	1229 State Street,	Santa Barbara, Cal.
1881	Dr. S. C. Ayres,	4 West 7th Street,	Cincinnati, Ohio.
1913	*Dr. Fred. D. Bailey,	260 Hancock Street,	Brooklyn, N. Y.
1907	Dr. C. H. Beard,	15 E. Washington Street,	Chicago, Ill.
1911	Dr. Arthur J. Bedell,	344 State Street,	Albany, N. Y.
1908	Dr. Geo. H. Bell,	40 East 41st Street,	New York, N. Y.
1905	*Dr. Jas. H. Bell,	Moore Building,	San Antonio, Texas.
1909	Dr. James Bordley, Jr.,	330 N. Charles Street,	Baltimore, Md.
1881	Dr. H. W. Bradford,		Wolfboro, N. H.
1908	Dr. E. V. L. Brown,	122 S. Michigan Blvd.,	Chicago, Ill.
1907	Dr. Wm. E. Bruner,	New England Bldg.,	Cleveland, Ohio.
1901	Dr. H. D. Bruns,	211 Camp Street,	New Orleans, La.
1910	*Dr. E. U. Buckman,	70 S. Franklin Street,	Wilkesbarre, Pa.
1906	Dr. W. K. Butler,	1207 M Street, N. W.,	Washington, D. C.
1906	Dr. W. Gordon M. Byers,	346 Mountain Street,	Montreal, Canada.
1909	Dr. L. W. Callan,	452 Fifth Avenue,	New York, N. Y.
1876	Dr. Peter A. Callan,	452 Fifth Avenue,	New York, N. Y.
1885	Dr. F. P. Capron,	118 Angell Street,	Providence, R. I.
O. M.	Dr. W. H. Carmalt,	261 St. Ronan Street,	New Haven, Conn.
1898	Dr. J. T. Carpenter,	2040 Chestnut Street,	Philadelphia, Pa.

Elected.	Name.	Residence.	Place.
1911	*Dr. James J. Carroll,	330 N. Charles Street,	Baltimore, Md.
1904	Dr. Burton Chance,	235 So. 13th Street,	Philadelphia, Pa.
1893	Dr. H. B. Chandler,	34½ Beacon Street,	Boston, Mass.
1904	Dr. J. W. Charles,	509 Humboldt Bldg.,	St. Louis, Mo.
1893	Dr. Wm. Cheatham,	305 W. Chestnut Street,	Louisville, Ky.
1893	Dr. F. E. Cheney,	64 Commonw'th Ave.,	Boston, Mass.
1898	Dr. J. H. Claiborne,	8 West 40th Street,	New York, N. Y.
1912	*Dr. E. W. Clap,	390 Commonw'th Ave.,	Boston, Mass.
1891	Dr. C. F. Clark,	188 East State Street,	Columbus, Ohio.
1903	Dr. Wm. G. Craig,	15 Fuller Building,	Springfield, Mass.
1912	*Dr. G. S. Crampton,	1700 Walnut Street,	Philadelphia, Pa.
1890	Dr. C. M. Culver,	36 Eagle Street,	Albany, N. Y.
1906	Dr. R. J. Curdy,	1134 Rialto Building,	Kansas City, Mo.
1902	Dr. Colman W. Cutler,	24 East 48th Street,	New York, N. Y.
1907	Dr. A. E. Davis,	50 West 37th Street,	New York, N. Y.
1878	Dr. W. S. Dennett,	8 East 49th Street,	New York, N. Y.
1909	Dr. George S. Derby,	7 Hereford Street,	Boston, Mass.
1874	Dr. Lewis S. Dixon,	232 Clarendon Street,	Boston, Mass.
1912	*Dr. Oscar Dodd,	77 E. Washington Street,	Chicago, Ill.
1902	Dr. Alexander Duane,	139 E. 37th Street,	New York, N. Y.
1908	Dr. J. L. Duncan,	Jenkins Bldg.,	Pittsburgh, Pa.
1912	*Dr. E. C. Ellett,	Exchange Building,	Memphis, Tenn.
1887	Dr. J. B. Emerson,	40 East 41st Street,	New York, N. Y.
1890	Dr. Arthur E. Ewing,	Metropolitan Bldg.,	St. Louis, Mo.
1896	Dr. T. H. Fenton,	1319 Spruce Street,	Philadelphia, Pa.
1915	*Dr. Carl Fisher,	102 S. Franklin Street,	Rochester, Minn.
1893	Dr. George F. Fiske,	25 E. Washington Street,	Chicago, Ill.
1909	Dr. M. L. Foster,	211 Centre Avenue,	New Rochelle, N. Y.
1904	Dr. W. H. Fox,	1826 Jefferson Place,	Washington, D. C.
1913	*Dr. Lee M. Francis,	575 Delaware Avenue,	Buffalo, N. Y.
1902	Dr. Percy Fridenberg,	38 West 59th Street,	New York, N. Y.
1894	Dr. H. Friedenwald,	1029 Madison Ave.,	Baltimore, Md.
1913	*Dr. H. G. Goldberg,	1925 Chestnut Street,	Philadelphia, Pa.
1895	Dr. G. M. Gould,	215 Atlantic Avenue,	Atlantic City, N. J.
1896	Dr. H. Y. Grant,	Falls View,	Ontario, Canada.
1908	Dr. L. S. Greene,	1624 I Street, N. W.,	Washington, D. C.
1905	Dr. Allen Greenwood,	101 Newbury Street,	Boston, Mass.
1914	*Dr. J. Milton Griscom,	1925 Chestnut St.,	Philadelphia, Pa.
1892	Dr. G. W. Hale,	210 Hitchcock Building,	Nashville, Tenn.
1895	Dr. Wm. D. Hall,	416 Marlborough Street,	Boston, Mass.
1887	Dr. H. F. Hansell,	1528 Walnut Street,	Philadelphia, Pa.
1901	Dr. Herbert Harlan,	516 Cathedral Street,	Baltimore, Md.
1889	Dr. David Harrower,	9 Elm Street,	Worcester, Mass.
1896	Dr. N. D. Harvey,	114 Waterman Street,	Providence, R. I.
1904	Dr. H. H. Haskell,	29 Commonwealth Ave.,	Boston, Mass.

Elected.	Name.	Residence.	Place.
1914	*Dr. Edw. B. Heckel,	Jenkins Building,	Pittsburgh, Pa.
1913	*Dr. Chas. R. Heed,	1402 Spruce Street,	Philadelphia, Pa.
1890	Dr. Neil J. Hepburn,	317 West 23d Street,	New York, N. Y.
1896	Dr. Ward Holden,	8 East 54th Street,	New York, N. Y.
1909	Dr. T. B. Holloway,	1819 Chestnut Street,	Philadelphia, Pa.
1906	Dr. C. R. Holmes,	8 East 8th Street,	Cincinnati, Ohio.
1883	Dr. E. E. Holt,	723 Congress Street,	Portland, Me.
1878	Dr. Lucien Howe,	520 Delaware Avenue,	Buffalo, N. Y.
1891	Dr. D. W. Hunter,	80 West 40th Street,	New York, N. Y.
1898	Dr. J. W. Ingalls,	874 Lafayette Avenue,	Brooklyn, N. Y.
1895	Dr. E. E. Jack,	215 Beacon Street,	Boston, Mass.
1885	Dr. Edward Jackson,	318 Majestic Building,	Denver, Col.
1912	*Dr. P. C. Jameson,	139 Montague Street,	Brooklyn, N. Y.
1892	Dr. W. B. Johnson,	170 Broadway,	Paterson, N. J.
1908	Dr. B. R. Kennon,	Taylor Building,	Norfolk, Va.
1901	Dr. H. W. Kilburn,	192 Marlborough Street,	Boston, Mass.
1903	Dr. Arnold Knapp,	10 East 54th Street,	New York, N. Y.
1889	Dr. Carl Koller,	681 Madison Avenue,	New York, N. Y.
1888	Dr. Chas. W. Kollock,	86 Wentworth Street,	Charleston, S. C.
1913	*Dr. Robert S. Lamb,	The Cecil,	Washington, D. C.
1891	Dr. W. E. Lambert,	112 East 35th Street,	New York, N. Y.
1902	Dr. W. B. Lancaster,	522 Commonw'lth Ave.,	Boston, Mass.
1912	Dr. H. M. Langdon,	2018 Chestnut Street,	Philadelphia, Pa.
1903	Dr. J. C. Lester,	432 Madison Avenue,	New York, N. Y.
1907	Dr. George F. Libby,	530 Metropolitan Bldg.,	Denver, Col.
1883	Dr. J. A. Lippincott,	Jenkins Building,	Pittsburgh, Pa.
1878	Dr. F. B. Loring,	1420 K Street,	Washington, D. C.
1909	Dr. R. G. Loring,	10 Arlington Street,	Boston, Mass.
1899	Dr. D. B. Lovell,	State Mutual Bldg.,	Worcester, Mass.
1914	*Dr. W. H. Lowell,	101 Newbury Street,	Boston, Mass.
1913	*Dr. A. L. Macleish,	1101 Brockman Bldg.,	Los Angeles, Cal.
1891	Dr. F. W. Marlow,	731 University Block,	Syracuse, N. Y.
1878	Dr. W. V. Marmion,	1211 13th Street, N. W.,	Washington, D. C.
1896	Dr. W. B. Marple,	46 W. 53d Street,	New York, N. Y.
1868	Dr. Arthur Mathewson,	Cosmos Club,	Washington, D. C.
1905	Dr. S. C. Maxson,	235 Genesee Street,	Utica, N. Y.
1907	Dr. C. H. May,	698 Madison Avenue,	New York, N. Y.
1887	Dr. Chas. H. McIlwaine,	320 Belleville Avenue,	Newark, N. J.
1874	Dr. C. S. Merrill,	27 S. Hawk Street,	Albany, N. Y.
1908	Dr. H. S. Miles,	417 State Street,	Bridgeport, Conn.
1888	Dr. B. L. Millikin,	1110 Euclid Avenue,	Cleveland, Ohio.
1883	Dr. J. Lancelot Minor,	Randolph Building,	Memphis, Tenn.
1881	Dr. W. F. Mittendorf,	140 Madison Avenue,	New York, N. Y.
1887	Dr. H. S. Oppenheimer,	276 Madison Avenue,	New York, N. Y.
1908	Dr. W. R. Parker,	David Whitney Bldg.,	Detroit, Mich.

Elected.	Name.	Residence.	Place.
1908	Dr. S. McA. Payne,	542 Fifth Avenue,	New York, N. Y.
1895	Dr. R. J. Phillips,	123 So. 39th Street,	Philadelphia, Pa.
1869	Dr. Thomas R. Pooley,	26 Trinity Street,	Newton, N. J.
1898	Dr. W. Campbell Posey,	2049 Chestnut Street,	Philadelphia, Pa.
1895	Dr. H. R. Price,	435 Clinton Avenue,	Brooklyn, N. Y.
1906	Dr. Brown Pusey,	7 West Madison Street,	Chicago, Ill.
1902	Dr. Walter L. Pyle,	1931 Chestnut Street,	Philadelphia, Pa.
1901	Dr. Alex. Quackenboss,	143 Newbury Street,	Boston, Mass.
1906	Dr. McCluney Radcliffe,	1906 Chestnut Street,	Philadelphia, Pa.
1885	Dr. Alex'r Randall,	1717 Locust Street,	Philadelphia, Pa.
1892	Dr. R. L. Randolph,	609 Park Avenue,	Baltimore, Md.
1891	Dr. J. M. Ray,	Atherton Bldg.,	Louisville, Ky.
1901	Dr. R. G. Reese,	50 West 52nd Street,	New York, N. Y.
1889	Dr. R. A. Reeve,	48 Bloor Street East,	Toronto, Canada.
1899	Dr. H. O. Reik,	506 Cathedral Street,	Baltimore, Md.
1912	*Dr. W. G. Reynolds,	1165 Dean Street,	Brooklyn, N. Y.
1884	Dr. Stephen O. Richey,	732 17th Street,	Washington, D. C.
1901	Dr. G. O. Ring,	2014 Chestnut Street,	Philadelphia, Pa.
1895	Dr. H. W. Ring,	185 Church Street,	New Haven, Conn.
1908	*Dr. J. Norman Risley,	36 7th Street,	New Bedford, Mass.
1880	Dr. Samuel D. Risley,	2018 Chestnut Street,	Philadelphia, Pa.
1887	Dr. Edmund C. Rivers,	1632 Welton Street,	Denver, Col.
1900	Dr. Wm. K. Rogers,	188 East State Street,	Columbus, Ohio.
1899	Dr. Dunbar Roy,	Gr. Opera House Blk.,	Atlanta, Ga.
1875	Dr. John D. Rushmore,	129 Montague Street,	Brooklyn, N. Y.
1880	Dr. Robert Sattler,	30, The Groton,	Cincinnati, Ohio.
1912	*Dr. A. C. Sautter,	1421 Locust Street,	Philadelphia, Pa.
1898	Dr. T. B. Schneideman,	1831 Chestnut Street,	Philadelphia, Pa.
1889	Dr. G. E. de Schweinitz,	1705 Walnut Street,	Philadelphia, Pa.
1895	Dr. P. N. K. Schwenk,	1417 North Broad St.,	Philadelphia, Pa.
1913	*Dr. C. E. G. Shannon,	1633 Spruce Street,	Philadelphia, Pa.
1905	Dr. John R. Shannon,	17 East 38th Street,	New York, N. Y.
1906	Dr. H. G. Sherman,	736 Rose Building,	Cleveland, Ohio.
1911	*Dr. J. F. Shoemaker,	1006 Carleton Bldg.,	St. Louis, Mo.
1902	Dr. Wm. A. Shoemaker,	1006 Carleton Bldg.,	St. Louis, Mo.
1902	Dr. Wm. T. Shoemaker,	109 South 20th Street,	Philadelphia, Pa.
1905	Dr. E. A. Shumway,	2007 Chestnut Street,	Philadelphia, Pa.
1912	Dr. E. T. Smith,	Conn. Mutual Bldg.,	Hartford, Conn.
1910	Dr. Albert C. Snell,	53 S. Fitzhugh St.,	Rochester, N. Y.
1909	Dr. F. M. Spalding,	390 Commonw'lth Ave.,	Boston, Mass.
1878	Dr. J. A. Spalding,	627 Congress Street,	Portland, Me.
1911	*Dr. Chas. N. Spratt,	900 Nicollet Avenue,	Minneapolis, Minn.
1884	Dr. Myles Standish,	72 St. James Avenue,	Boston, Mass.
1873	Dr. George T. Stevens,	40 East 41st Street,	New York, N. Y.

Elected.	Name.	Residence.	Place.
1903	Dr. Edw. Stieren,	Westinghouse Bldg.,	Pittsburgh, Pa.
1887	Dr. T. Y. Sutphen,	992 Broad Street,	Newark, N. J.
1900	Dr. Wm. M. Sweet,	1205 Spruce Street,	Philadelphia, Pa.
1888	Dr. Lewis H. Taylor,	83 So. Franklin Street,	Wilkesbarre, Pa.
1880	Dr. Samuel Theobald,	Cathedral & How'd Sts.,	Baltimore, Md.
1893	Dr. Ch. Hermon Thomas,	3634 Chestnut Street,	Philadelphia, Pa.
1898	Dr. A. G. Thomson,	Stock Exchange Bldg.,	Philadelphia, Pa.
1903	Dr. Edgar S. Thomson,	405 Park Avenue,	New York, N. Y.
1898	Dr. Jas. Thorington,	2031 Chestnut Street,	Philadelphia, Pa.
1908	Dr. Andrew Timberman,	112 East Broad Street,	Columbus, Ohio.
1911	Dr. Fred. Tooke,	368 Mountain Street,	Montreal, Canada.
1912	*Dr. E. Török,	8 East 54th Street,	New York, N. Y.
1906	Dr. H. H. Tyson,	47 West 51st Street,	New York, N. Y.
1898	Dr. C. A. Veasey,	Paulsen Building,	Spokane, Wash.
1905	Dr. F. H. Verhoeff,	101 Newbury Street,	Boston, Mass.
1903	Dr. F. L. Waite,	68 Pratt Street,	Hartford, Conn.
1873	Dr. David Webster,	24 East 48th Street,	New York, N. Y.
1893	Dr. J. E. Weeks,	46 East 57th Street,	New York, N. Y.
1914	*Dr. Walter B. Weidler,	62 West 58th Street,	New York, N. Y.
1900	Dr. C. D. Wescott,	22 E. Washington Street,	Chicago, Ill.
1891	Dr. Edward Wheelock,	26 Gibbs Street,	Rochester, N. Y.
1892	Dr. J. A. White,	200 E. Franklin Street,	Richmond, Va.
1895	Dr. W. H. Wilder,	1417 Peoples Gas Bldg.,	Chicago, Ill.
1910	Dr. Carl Williams,	School L. & Greene St.,	Philadelphia, Pa.
1874	Dr. Chas. H. Williams,	1069 Boylston Street,	Boston, Mass.
1912	Dr. Edw. R. Williams,	1069 Boylston Street,	Boston, Mass.
1893	Dr. Wm. H. Wilmer,	1610 I Street, N. W.,	Washington, D. C.
1888	Dr. F. M. Wilson,	834 Myrtle Avenue,	Bridgeport, Conn.
1906	Dr. J. Scott Wood,	172 Sixth Avenue,	Brooklyn, N. Y.
1900	Dr. Hiram Woods,	842 Park Avenue,	Baltimore, Md.
1915	*Dr. H. W. Wootton,	319 Lexington Avenue,	New York, N. Y.
1881	Dr. J. P. Worrell,	20 South 7th Street,	Terre Haute, Ind.
1905	Dr. Wm. Zentmayer,	1819 Spruce Street,	Philadelphia, Pa.
1903	Dr. S. Lewis Ziegler,	1625 Walnut Street,	Philadelphia, Pa.

Total 193

HONORARY MEMBERS.

O. M.	Dr. B. Joy Jeffries,	15 Chestnut Street,	Boston, Mass.
1868	Dr. J. S. Prout,	152 Putnam Avenue,	Brooklyn, N. Y.
O. M.	Dr. F. P. Sprague,	229 Commonw'lth Ave.,	Boston, Mass.

Whole number 196

Dr. Hasket Derby.

NECROLOGY.

DR. HASKET DERBY.

MYLES STANDISH, M.D.,
Boston, Mass.

Dr. Hasket Derby was born in Boston, June 29, 1835. He came from a family well known and influential in Salem, Mass., for generations. He was graduated from Amherst College in 1855, and from the Harvard Medical School in 1858. He served in the Massachusetts General Hospital as House Pupil for one year, and immediately went abroad, where he spent the succeeding three years in the study of medicine. Eighteen months of this time was devoted to general medicine, after which he took up special study of the eye under von Arlt and Jaeger in Vienna, then under von Graefe in Berlin. He worked for a considerable period with Donders in Utrecht.

He carefully studied the practice of Bowman, Critchett, and Hutchinson in London, and that of Desmarres and Sichel in Paris. Of all these eminent men who were developing ophthalmology at this important era, immediately after the invention of the ophthalmoscope by Helmholtz, the one who exercised the greatest influence over the young student was von Graefe. He was an enthusiastic admirer of that great ophthalmologist, and throughout life would occasionally quote his opinions. He placed a bust of von Graefe prominently in his consulting-room at the Massachusetts Eye and Ear Infirmary.

Dr. Derby had all the qualities which make a great practitioner of medicine. Prompt, accurate, observing, acute, and resourceful, he was an excellent diagnostician, and commanded the confidence of his patients and colleagues.

He was a man of positive opinions and forceful personality, and, as Dr. David W. Cheever has written of him: "What he knew, he knew he knew, and there was no latitude allowed"—an illuminatingly descriptive phrase. His associates, however, would add, he not only knew that he knew, but also acted courageously upon that knowledge. For example, he was convinced that the nausea and excitement which follow an ether anesthesia were harmful to an eye as widely opened as is the case for the extraction of the lens, and therefore operated, before the discovery of local anesthetics, upon all cases of senile cataract without any anesthetic whatever. Such was his control of his patients that those upon whom he was obliged to give up the method and administer ether were surprisingly few. He was a good operator, and did a large amount of operative work.

He was successful from the first, acquiring a large private practice which followed him throughout his professional career. His patients had great confidence in him, and justly, for he added to those qualities which made him professionally able a conscientiousness which kept him sternly up to his duty under all circumstances. This quality also made him a man of strong religious convictions, who never wavered in his loyalty to his church and faith.

Dr. Derby was kind to his patients, and generous with his time and skill to the poor. A well-known public-spirited citizen in the city in which he lived all his life, he was a trustee in the Children's Institutions Department of the City of Boston, and often associated with movements for the good of the community. He organized the New England Ophthalmological Society, and was its first president.

He was one of eight ophthalmologists who met in the

office of Dr. Henry D. Noyes, in New York, in January, 1864, and organized the American Ophthalmological Society. Subsequently he was for five years its president.

Fond of the English classics, with ability to read French and German with facility, he was of distinctly scholarly habit, yet his most active interest was always devoted to his public clinics.

He was Ophthalmic Surgeon in the Massachusetts Charitable Eye and Ear Infirmary for thirty years. Among his colleagues on the Board of Surgeons he was ever active and aggressive in forwarding the interests of the institution as he saw them. His juniors found him a strict disciplinarian, but one who always gave full credit for work well done, and who never missed an opportunity to forward their interests in the hospital or to assist them in establishing themselves in private practice. Prompt himself, arriving on the minute he designated, he expected his assistants to be equally efficient and active. The great clinic at the Infirmary was not able to absorb all his energy, and coincidentally he established the eye clinic at the Carney Hospital, and was for five years the only attending surgeon.

Dr. Derby was a fellow of the Massachusetts Medical Society from 1862 until his death, and for many years one of its councillors. For several years he was Lecturer on Ophthalmology at the Harvard Medical School. He served under the Sanitary Commission in the latter part of the Civil War.

As a medical writer he was forceful and practical, and a large proportion of his articles will be found in the Transactions of this Society.

He gave up practice four years before his death, and lived in retirement at Milton, Massachusetts.

He will be remembered with admiration by all who knew him, and especially by those who had the good fortune to be his assistants during his active career.

Dr. Derby died August 21, 1914, and is survived by his widow, Sarah Mason, of Boston, to whom he was married in 1868, and by six children, Eloise and Dr. George S., of Boston; S. Hasket, of San Francisco; Robert M. and Augustine, of New York; and Arthur, of New Orleans.

DR. GEORGE STRAWBRIDGE.

S. LEWIS ZIEGLER, M.D.,
Philadelphia.

By the death, on June 28, 1914, of Dr. George Strawbridge, the American Ophthalmological Society has lost one of its early and most distinguished members, and the Wills Eye Hospital has been deprived of an Emeritus Surgeon who, in his day, was one of the most brilliant of the active surgical staff that helped to establish the world-wide reputation of that institution during the early seventies and the decade following.

Dr. Strawbridge was born in Philadelphia on October 18, 1844. He was the son of George Strawbridge and Jane B. West. He acquired his preliminary education at the Germantown Academy, Philadelphia, and was graduated from the University of Pennsylvania in 1863 with the degree of Bachelor of Arts. In 1866 he graduated from the Medical Department of the same institution, and was made a Doctor of Medicine. He was then elected a Resident Physician to the Episcopal Hospital, where he remained for one year. He was one of the first of a small coterie of American physicians to go abroad for the avowed study of ophthalmology, which at that time was just beginning to be differentiated as a special field of medicine, and was being developed in the great hospitals of Europe. He also planned to include otology in his field of study.

Dr. George Strawbridge.

Having made up his mind to go abroad, he naturally explained his plans to his preceptor, Professor Joseph Carson, who promptly retorted, "A fool and his money are soon parted." In relating this incident he naïvely added, "With this encouragement I sailed for Europe the next day." He went abroad in May, 1867, and spent three years in study under the brilliant masters of that time. In Vienna he associated himself with Arlt, Jaeger, and Stellwag, and was thus enabled to observe many of the interesting cases which they afterward placed on record. He was especially fortunate in being able to study, at first hand, many of the ophthalmoscopic pictures which have been immortalized by Jaeger in his "Atlas." He studied for some time with von Graefe in Berlin, and often related to his friends the interesting experiences which he enjoyed while there. Later he took a course with Donders and Snellen in Utrecht, and with Helmholtz in Heidelberg. He also spent several months with Bowman and Critchett in London. He was thus imbibing his knowledge at the very fountain-head of modern ophthalmology.

He returned home in 1870, and at once associated himself with the Philadelphia Dispensary, establishing an eye dispensary on Seventh Street, which attracted 700 new patients during the first year. During the same year he was appointed Clinical Lecturer on Diseases of the Eye and Ear at the University of Pennsylvania, which position he held until the division of those specialties into two chairs, in 1873, when he was made Clinical Professor of Diseases of the Ear, the professorship of Diseases of the Eye falling to the lot of his associate, Dr. William F. Norris. He held that position from that time until he resigned in 1890. He was always a forceful and interesting lecturer, and every student in his ward class carried away with him some impressive and practical point in otology.

In 1872 he was elected Ophthalmic Surgeon to the Pres-

byterian Hospital, which position he held until he retired in 1913, when he was made Consulting Ophthalmic Surgeon.

In 1873 there was a reorganization of the Surgical Staff of the Wills Eye Hospital and he was elected an Attending Surgeon. Owing to failing health he resigned this position in 1890, and was elected Emeritus Surgeon. This brilliant service, therefore, covered a period of seventeen years.

Notwithstanding his many other professional activities he founded, in 1875, the Pennsylvania Eye and Ear Infirmary, which he maintained as a private free dispensary at Thirteenth and Chestnut Streets, Philadelphia, up to the day of his death. He carried this work on much as he had seen it done under similar auspices during his sojourn in Europe.

Dr. Strawbridge was married on June 5, 1873, to Miss Alice Welsh, daughter of John Welsh, a well-known banker of his time. She proved to be a good helpmeet, and bore him four children, two sons and two daughters, all of whom survive him.

Dr. Strawbridge had one of the largest and most lucrative practices of his day. He was a good business man, and accumulated a large fortune through judicious real-estate investments. He was always kind to those around him, and most generous in all his dealings. Although he sometimes spoke cynically of human affairs as he saw them, he was a true optimist at heart. He was always genial and often jovial. He invariably inspired hope and confidence in his patients. He never lost his temper or even became rattled under the most trying circumstances. If the patient lost his nerve under the stress of operation, he never gave sharp orders. He simply patted him on the back and encouraged him by saying "you are doing fine," when he was possibly squeezing out the lens and part of the vitreous. It is related that in the pre-cocain days, when eyes were trained for operation by daily tapping the cornea, he allowed

an excited patient to jump out of bed and run around the ward with the knife sticking through the eye, after which performance he calmly asked the patient to lie down again if he had finished with his exercise.

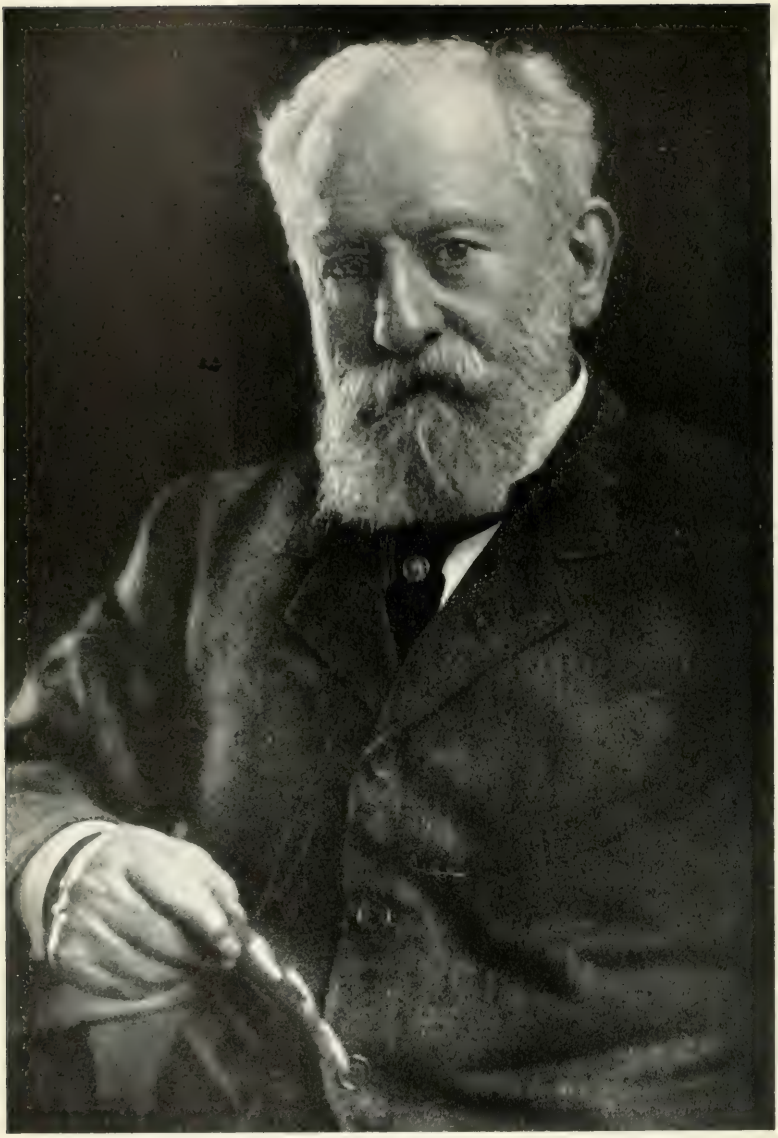
The writer was House Surgeon at the Wills Eye Hospital for two years, from 1887 to 1889, during which period cocain was first being used and antiseptic surgery was in its infancy; he can, therefore, speak of many things from the standpoint of personal association. Dr. Strawbridge was a great operator, a good clinician, and an expert in the use of the ophthalmoscope. He was not wholly a therapeutic nihilist, but he had no great faith in drugs, although pleased and happy when his therapy proved successful. He seldom used mydriatics, and even persisted in "non-mydriatic refraction," as he called it. His expert use of the ophthalmoscope enabled him to secure a fairly accurate refraction. In a series of cases requiring refraction that he referred to the writer he wagered that both methods would give the same result. It was demonstrated, however, that the so-called "Philadelphia Method" of refraction, under a cycloplegic, was the more accurate. He accepted antisepsis reluctantly, but finally became an enthusiastic supporter of the method. He first used mercury biniodid in Panas' solution, but this produced in several cases a persistent and puzzling quiet iritis, which led him to adopt other measures of a less irritating character.

He was essentially a surgeon, and performed more operations than his colleagues, chiefly because of his personal following and because of the large number of cases referred to him by his private infirmary. He was a rapid and accurate operator. His record in cataract operations was excellent. He made the high corneal incision, which was a counterpart of the more modern so-called Smith-Indian incision. He used the narrow Graefe knife, and turned the blade-edge sharply forward at a right angle with the

cornea as he emerged, thus leaving a square-edged shoulder. As his points of puncture and counterpuncture were outside the limbus, there was more gaping of the wound than was customary with the incisions of other operators. Occasionally a small fragment of tissue which had lodged in the angle of the wound, or a rubbing together of the square-shouldered wound edges, would cause delayed union. When such a condition became evident to him, he removed all the bandages and allowed the lid friction to stimulate the healing process. This was usually followed by prompt union of the wound.

His plastic work was excellent and usually successful. During the final year of the writer's service as House Surgeon all his efforts and ingenuity were directed toward devising a successful method for making an artificial pupil. He tried von Graefe's knife à la Cheselden, he used Hays' knife in many ways, he performed de Wecker's iritoectomy, he devised a pair of very small scissors for puncturing and incising the iritic membrane, and finally, in 1888, he began the use of von Hippel's trephine, which he employed to transplant the cornea, to trephine the occluding iritic membrane, and to trephine the sclera. He usually covered the scleral wound by replacing the conjunctiva, in order that it might become a transparent visual membrane. He also performed scleral trephining for glaucoma, and in one instance for retinal detachment, some of which cases were reported by him. He should, therefore, receive credit for being a pioneer in this work, even though his technic was not the same as at present employed. The Transactions of this Society contain the most of his papers describing his various operations. His many attempts to secure a successful artificial pupil inspired the writer to similar efforts, and led to the discovery of the V-shaped iridotomy, which at a later date was practised on one of the same patients.

During the latter part of the year 1899 Dr. Strawbridge noticed that his judgment as to the counterpuncture in



E. Gruening

cataract incisions was not good, since his knife sometimes emerged too far in the sclera. A consultation with Drs. Heyl, Harlan, and Knapp revealed a doubtful melanosarcoma of the choroid in the left eye. An early enucleation was performed. A microscopic examination by Dr. Knapp showed a fibrous character that led to a favorable prognosis of non-malignancy. This prognosis proved correct, as he lived for a quarter of a century, but he never quite recovered his normal poise, and his activities were considerably limited for many years. He resigned from the Wills Eye Hospital, from the University of Pennsylvania, and from most of his medical societies in 1890. He continued to serve his own dispensary and to maintain a limited office practice up to the time of his death.

He finally succumbed to what appeared to be a recurrence of the original trouble, which brought hepatic involvement and metastases to other parts of the body. His hopeful mental attitude persisted to the very end of a life that was active, honorable, and most useful to mankind.

DR. EMIL GRUENING.

WILBUR B. MARPLE, M.D.,
New York.

Dr. Emil Gruening, one of the ablest and most prominent members of our Society, died on May 30, 1914.

He was born in Hohensalza, not far from Thorn, in East Prussia, on the second day of October, 1842. He graduated in the gymnasium, and it was his first intention to take up teaching as a profession.

At the age of twenty he came to the United States, and landed here without friends, and with no one to assist him. He was fond of languages, and tutored in Latin and French in the families of several New York people, among them in

the family of Dr. Willard Parker, and it was Dr. Parker who advised the young tutor to study medicine.

With the funds thus obtained he began his medical course at the College of Physicians and Surgeons in 1864. He pursued his studies here only a year, however, when his devotion to his newly adopted country led him to enlist in the army. He had quite a circle of German friends in Hoboken, and these all got together and enlisted in the same company, so as to be together. It was thus that he went to the war in the Seventh New Jersey Volunteer Infantry instead of in a New York regiment. I have often heard him tell of the hardships and privations which he had to undergo, and the battles in which he took part in the campaign in Virginia, where he was present at Appomattox when Lee surrendered.

He was mustered out at the end of his service, returned to New York, and continued his medical studies, obtaining his degree in 1867.

The next three years he spent abroad, studying in London, Paris, and Berlin, working with von Graefe and others.

Returning in 1870 to New York, he was soon afterward appointed on the staff of the New York Ophthalmic and Aural Institute. Here he was associated with Dr. Herman Knapp, and afterward he assisted the latter in his private office. He always had the greatest respect and admiration for this great master.

In 1878 he became Ophthalmic Surgeon at the New York Eye and Ear Infirmary, a position he held until 1912, when he became consultant.

In 1879 he became Attending Ophthalmologist to the German Hospital, and in 1903 he resigned and was appointed consultant.

In 1884 he was appointed Ophthalmic Surgeon to Mt. Sinai Hospital, a position he held until 1909, when he resigned and was appointed consultant.

He became Professor of Ophthalmology at the New York Polyclinic Medical School in 1881, a position which he held for thirteen years. He was also consulting surgeon to the New York Infirmary for Women and Children.

He was a member of all the prominent special societies, having become a member of the American Ophthalmological Society in 1875, of which he was president in 1910. He became a member of the New York Ophthalmological Society in 1870, and was elected its president in 1886.

He was a member of the Section on Ophthalmology of the New York Academy of Medicine, and for two years was its chairman.

He was one of the Directors of the New York Eye and Ear Infirmary, a member of the New York Academy of Medicine, the County Medical Society, Medical Society of Greater New York, German Medical Society, New York Otological Society, American Otological Society (its President in 1903), New York Physicians' Mutual Aid Association, and President of the Society for the Relief of Widows and Orphans of Medical Men.

His contributions to the literature of ophthalmology and otology were numerous and able. In the System of Diseases of the Eye by Norris and Oliver he contributed the chapter on "Injuries of the Eye." It would be impractical in the space allotted to give the titles of all his contributions. Among the most important were papers read before this Society—"The Use of the Curette in Inveterate Pannus" (1899); "Treatment of Ptosis by Partial Resection of the Tarsal Cartilage" (1902); "The Operative Treatment of Divergent Strabismus" (1891). Other papers were: "Methyl Alcohol Amblyopia," *Arch. für Augenheilk.*, v, lxix; "On Quinin Amaurosis," Reports N. Y. Eye and Ear Infirmary (1897); "Reflex Ocular Symptoms in Nasal Affections," *Medical Record* (1886).

It was the writer's privilege to have been associated in-

timately with Dr. Gruening for almost twenty-five years before his death, and for ten of these years he was his Chief of Clinic at the New York Eye and Ear Infirmary. This long association proved to be an invaluable training in ophthalmology. Dr. Gruening was an ideal teacher, and one learned very much from him, and at the same time his industry and enthusiastic interest in his work were a constant inspiration to all who came within the sphere of his influence.

He was always most interested in the young men who worked or studied with him, and endeavored to help them in their work in every possible way. Scattered over the length and breadth of our land are many of our best men who regarded Dr. Gruening with the greatest affection and gratitude for the assistance and kindness he had shown them at some time in their careers.

Dr. Gruening was a learned man, not only in medicine, but in all departments of knowledge, and there were few subjects, even outside of medicine, on which he could not converse intelligently. In this respect he was a refreshing contrast to the many who may be learned in the things concerning their special work, but are interested in and know nothing of anything else. Though a very busy man, he was an indefatigable reader, and possessed a remarkable memory, so that I was constantly surprised at his great learning; at his familiarity with almost everything that was new and good in all departments of medical literature. He was very fond of music, and was a member of the Sunday Musical Club, and attended their weekly sessions, where the choicest chamber music was interpreted by the best professional performers.

His knowledge of the best literature of all times was most intimate, and often have I heard him make apropos quotations from both ancient and modern classics.

His family life and interest in his children and in their

education, not a little of which they imbibed from his own teachings, were most charming. His widow and four children survive him.

Dr. Gruening was in active practice forty-three years, specializing in both ophthalmology and otology, and achieved the most distinguished success, whether measured by his very large and private clientele or by the standing and influence which he attained among his colleagues.

He was a most skilful diagnostician. For many years he was ophthalmic surgeon to two of our largest general hospitals. These positions brought him into intimate association with the best men in all departments of medicine, so that he was kept constantly in touch with the work of all specialties. The result was that he had a much broader medical horizon than those whose work was limited to that seen in a special eye hospital, and this great advantage possessed by Dr. Gruening was in constant evidence.

He was most honorable in all his dealings, with both patients and colleagues. On one occasion he described at one of our societies a case in which he had operated on both eyes of a patient, and in whom the second eye became infected. Some one asked him if he had sterilized the instruments between the first and second operation. I will never forget that occasion. Not one of his hearers was present at the operation, so he could easily have answered the question in the affirmative, and no one would have known the difference. But when the question was asked him, he stood thinking a moment, then turned and replied without hesitation, "I do not think I did!"

He was an exceptionally skilful operator, and this skill he maintained up to the end. About three months before his death I well remember one of the house surgeons at the Infirmary saying one afternoon, "I have just seen Dr. Gruening do as beautiful a cataract extraction as I have ever seen," and he was then over seventy years old.

He was a most careful clinical observer, an instance of which is his observation that, where a small foreign body penetrates the globe through the cornea, if there is no iris prolapse, the foreign body will most surely be found in the globe. If, however, there is a prolapse, the foreign body must have had a much less velocity, and so might not have entered the globe at all. As this observation was made long before the days of the Roentgen-ray, it was at that time one of much importance, whose accuracy I have many times had the opportunity of corroborating.

He was a regular attendant at the meetings of our special societies, and his contributions to the discussions based on his learning and large experience were always most instructive, and I am sure that no one is as much missed to-day in these societies as is Dr. Gruening.

He had a good deal of dry humor. On one occasion, when a paper was read at one of our meetings on what seemed to him a rather trite subject, he remarked that the paper reminded him of the occasion when he had listened, after hearing it many times previously, to the opera "Die Meistersinger"; he had discovered new beauties in the opera which he had missed in the previous hearings!

His interest and alertness in his work continued up to the end. He contributed to the Transactions of this Society in 1912 his experience in twenty-one cases of the Lagrange operation. Two months before his death he presented, with the history, a patient, previously seen by a confrère, who made the diagnosis of atrophy of the optic nerves. When Dr. Gruening saw the patient he took the visual fields, found he had bitemporal hemianopsia, and made the diagnosis of apophysis disease. The patient was operated on, and great improvement of vision followed.

Dr. Gruening died literally "in the harness." Not more than two weeks previously he attended a meeting of the surgeons of the Infirmary. He was somewhat lame, walked

Dr. M. Hayward Post.

with a cane, and thought he had sciatica, but this subsequently proved to be endarteritis obliterans associated with arteriosclerosis.

He was a rare and a great man. To have known him at all was a privilege, but doubly fortunate were those who were permitted to have known him at all intimately. These loved and venerated him, and to them his loss has been irreparable.

DR. M. HAYWARD POST.

A. E. EWING, M.D., AND J. W. CHARLES, M.D.,
St. Louis, Mo.

In the restlessness that characterizes modern civilization, particularly in the United States, the idea has rather become established, with each rising generation, that a young man must change his place of abode from that in which he was reared if he would rise to eminence. To remain in the region of his birth, live down the almost irresistible weight of this impression, and through the power of his own great character to earn the sincerest esteem of the playmates of his childhood and of the community of his youth, and along with this to rise gradually in his profession to the highest office of the most advanced society of his chosen profession, means genius and stamina. In the person of our friend, Dr. Post, these were combined with a personal charm that evoked the deepest respect and admiration. He was born in St. Louis, March 31, 1851, his father being Dr. Truman Marcellus Post, the pioneer minister in the Congregational Church west of the Mississippi River, a man of great eloquence and wisdom; and his mother was of the strong-minded, gentle type that characterized the noble women of this period. Early in his life the son connected himself

with the First Congregational Church, in which he later became one of the chief advisers, and at the time of his death he was both a Deacon and a member of its Board of Trustees.

His preliminary education was obtained in the public schools, in Smith Academy, which is the preparatory school of Washington University, and in the University itself, from which he received the degree of A. B. in 1872, being the honor man of his class. With the idea in view of fixing and retaining the education for which he had labored, as well as for the general broadening of his faculties, he became a teacher in the public schools of St. Louis. At the time of his resignation for the purpose of studying medicine he was Principal of the Blow School in Carondelet. In 1877 he graduated from the St. Louis Medical College, the leading medical institution west of the Mississippi, now the Medical Department of Washington University, and became an intern in the St. Louis City Hospital. After this internship he entered general practice in St. Louis, numbering among his friends and counselors the eminent surgeons Hodgen and Gregory, and the general practitioners Baumgarten and Fischel, but he leaned to surgery, and was much in the company of Hodgen as an extra assistant, until he became the chosen assistant of Dr. John Green in ophthalmology. From this assistantship he went directly to Europe, where he took special postgraduate courses with Donders and Snellen in Utrecht, and instruction in ophthalmic surgery under Nettleship in London. To him these three names were ophthalmic Penates, so much did he value the knowledge that he acquired while with them.

On his return from Europe he became associated with Dr. Green in the practice of ophthalmology, an association of friendship, respect, and trust that was broken only by the death of Dr. Green in 1913—a life association of two great men, great minds, scholars, gentlemen, each deeply in-

terested in maintaining the noblest civic and professional ideals. For many years a free ophthalmic clinic was conducted by them at St. Luke's Hospital, and also another free clinic was conducted by Dr. Post at the Protestant Hospital, until both were merged into the present ophthalmic clinic of Washington University.

For a number of years Dr. Post was ophthalmic consultant to the St. Louis City Hospital, the St. Louis Female Hospital, and the St. Louis Eye, Ear, Nose, and Throat Infirmary, and an active member of the Medical Board, as well as ophthalmologist to the Protestant Hospital, the Missouri Baptist Sanitarium, and to the St. Luke's Hospital. He was a member of the American Medical Association, the American Ophthalmological Society, the American College of Surgeons, the American Academy of Medicine, the St. Louis Academy of Science, the St. Louis Medical Society, the St. Louis Ophthalmological Society, of which he was vice-president at his death, and the Ophthalmic Section of the St. Louis Medical Society, of which he was Chairman several years ago. In 1912 he was Chairman of the Washington University Association, and in 1914 was elected honorary member of the Phi Beta Kappa Society. In 1887 he was elected a member of the American Ophthalmological Society, was chosen as its vice-president in 1913, and as its president in 1914, an honor that he appreciated as the greatest in the gift of the special branch of his profession.

Although burdened with a large private practice, which to the small mind means comfort and luxury, Dr. Post never lost sight of the interest of the community, or of his duty in lending it his knowledge and his energy to better its condition along ophthalmic lines. An active member of a large church in which were many wealthy members, he did not hesitate to let them know in his quiet, unobtrusive way what was needed to help the blind unfortunates of the State

and the city, and much was contributed by them to aid him in his charitable work, the extent of which was known to only a few of us who were immediately associated with him. Among the good seeds thus sown, one has developed into a future legacy for Washington University that will forever continue in a large way the clinical work in ophthalmology which he and Dr. Green so carefully maintained for so many years in connection with the Protestant Hospital and St. Luke's Hospital. In the State of Missouri his work is graven into a law, known as the Ophthalmia Neonatorum Law, for the protection of infants suffering with this disease against carelessness and negligence. Also his unselfish magnanimity is further recorded in the annals of the State by twenty-four years of service as consultant to the Missouri School for the Blind, and as a member of the Board of Managers.

As he himself, in his earliest medical career, had been the disciple of the great professional ability of the community, so he became in his later years counsel and friend to the younger men in a great and masterful manner, free from the tinge of self-superiority or imperiousness, a manner that drew those who were his assistants to him and made them life confidants. The same attitude was always maintained toward those who trusted him as patients: he was their friend, cheered them in their misery, and inspired hope when life to them has become hopeless. Amid his immediate circle of friends, his genial personality, together with his intelligent prudence and the soundness of his advice, combined with the kindly spirit in which they were exercised, ripened friendship into a deep and permanent respect and affection.

In the spring of 1914, his sixty-third year, he was stricken with pneumonia, which involved the heart, and ended in a fatal attack of angina pectoris September 1, 1914. He is survived by his widow and four sons, two of whom are ophthalmologists.

BIBLIOGRAPHY OF THE WRITINGS OF M. HAYWARD POST, M.D.

(*American Journal of Ophthalmology.*)

1885. Vol. II, page 315—A Case of Transplantation of Two Skin Flaps without Pedicle.
1887. Vol. IV, page 163—Rupture of the Optic Nerve at the Chiasm.
1890. Vol. VII, page 148—Removal of a Piece of Steel from the Vitreous Body with Recovery of Perfect Vision.
1900. Vol. XVII—Report of a Case of Choroidal Sarcoma in a Syphilitic.
1905. Vol. XXII, No. 3, page 86—An Experience with Staining the Skin with Argyrol.
1907. Vol. XXIV, No. 5, page 129—Detachment of the Retina.
1908. Vol. XXV, No. 5, page 129—A New Knife for Dividing Membranes Obstructing the Pupil after Cataract Operations.
1908. Vol. XXV, No. 12, Page 356—A Successful Operation on a Cataract Complicated by a Dacryocystitis.
1909. Vol. XXVI, No. 2—Report of Needling of a Cataract in a Patient Thirty-five and another Thirty-seven Years of Age.
1910. Vol. XXVII, No. 4, page 97—Report of Several Cases of Neuropathic Keratitis.
1910. Vol. XXVII, No. 9, page 257—Prevention of Blindness from Ophthalmia Neonatorum.

(*Transactions of the American Ophthalmological Society.*)

1911. Vol. XII, Part III, page 759—A Case of Gonorrheal Conjunctivitis with Arthritis Preceding Conjunctival Discharge by Four Days.

(*American Journal of Ophthalmology.*)

1911. Vol. XXVIII, No. 12, page 353—Prevention of Ophthalmia Neonatorum.
1912. Vol. XXIX, No. 2, page 40—(Post and Alt)—Bony Tumor of Orbit; Microscopic Examination.
1913. Vol. XXX, No. 6, Page 161—Optic Neuritis Dependent upon Inflammation in the Ethmoid Cells.

MINUTES OF THE PROCEEDINGS.

FIFTY-FIRST ANNUAL MEETING.

Eastern Point, New London, Conn., *July 6, 1915.*

The Fifty-first Annual Meeting of the Society was called to order at 9 A. M. by the Vice-President, Dr. George E. de Schweinitz, of Philadelphia.

Dr. de Schweinitz: In accordance with the provisions of Section 2, Article III, of the By-Laws, I have the honor to request your indulgence in that I shall assume the duties of Presiding Officer at this meeting. Of Dr. Post's work and worth I need not speak. Due notice of them will appear in our Transactions. It is, however, fitting and proper that some mention shall now be made of our late President and of his work and worth to which I have referred, and take the form of a minute to be spread on our records.

Dr. Weeks: I should like to make a motion such as you have suggested with regard to our late associate and President of this Society, Dr. Post. I move that a committee be appointed to prepare a minute to be spread upon our Minutes.

This motion was seconded and carried, and the Chair appointed on this Committee Dr. John E. Weeks, Dr. A. E. Ewing, and Dr. S. D. Risley.

The following Committee was appointed:

Auditing Committee: Dr. W. B. Lancaster.

The scientific program was then taken up, and the following papers were read and discussed:

1. "A Review of the Glaucoma Question, with Special Reference to Newer Conclusions of its Etiology and De-

partures of its Surgical Treatment." By Robert Sattler, M.D., Cincinnati, Ohio.

Discussed by Drs. Koller, Marple, Weeks, Cutler, Wilmer, and Sattler.

2. "A Report of Several Cases of Chronic Simple Glaucoma Treated by Iridotasis." By David Harrower, M.D., Worcester, Mass.

Discussed by Drs. Verhoeff, Sattler, P. A. Callan, Koller, Lovell, H. Woods, O'Connor, Cahill, and Harrower.

3. "Tenotomy of the Inferior Oblique Muscle." By Wm. Campbell Posey, M.D., Philadelphia.

Discussed by Drs. Duane, Zentmayer, Shumway, Weeks, and Posey.

4. "Rosacea Keratitis and Certain Other Forms of Marginal Keratitis, Neuropathic in Origin. Treatment by Pericorneal Neurotomy." By F. H. Verhoeff, M.D., Boston, Mass.

Discussed by Drs. Holloway and Verhoeff.

5. "Some Experiences in Enucleation Under Local Anesthesia." By William M. Sweet, M.D., Philadelphia.

Discussed by Drs. Lancaster and Butler.

6. "Massive Granuloma of the Sclera (Brawny Scleritis)." By George S. Derby, M.D., Boston, Mass. Pathologic examination by F. H. Verhoeff, M.D., Boston, Mass.

7. "Salvarsan in Interstitial Keratitis and in Optic Nerve Lesions." By Arnold Knapp, M.D., New York City.

Discussed by Drs. Bordley, Koller, P. A. Callan, Marple, Randolph, Derby, J. R. Shannon, Smith, Holloway, Ziegler, Wilder, de Schweinitz, and Knapp.

The following physicians were introduced as guests of the Society, and invited to take part in the discussions: Drs. J. H. Haughey, Aurora, Nebraska; D. F. O'Connor, Worcester, Mass.; Clifford B. Walker, Boston, Mass.; J. W. Cahill, Worcester, Mass.; A. C. Woods, Boston, Mass.; and Thos. A. Woodruff, Chicago, Ill.

AFTERNOON SESSION, JULY 6TH.

The meeting was called to order at 2.15 o'clock, Dr. de Schweinitz, presiding.

Exhibition of Instruments and Apparatus.

8. "The Ives-Langdon Photometer." By H. Maxwell Langdon, M.D., Philadelphia.

Discussed by Dr. C. H. Williams.

9. "A Review of the History of the Electric Ophthalmoscope, with a Demonstration of a New Ophthalmoscope, which Consists of a Rearrangement of the Parts of Antecedent Instruments." By J. Herbert Claiborne, M.D., New York City.

Discussed by Drs. Marple, Callan, Ziegler, and Claiborne.

10. "A Simple Modification of the Berger Loupe." By Edward A. Shumway, M.D., Philadelphia.

Discussed by Drs. Marple and Shumway.

11. "Demonstration of Nagel's Anomaloscope for Testing Color Vision." By Chas. H. Williams, M.D., Boston, Mass.

12. "The May Electric Ophthalmoscope." By C. H. May, M.D., New York City.

Discussed by Drs. Ziegler, Verhoeff, and May.

13. "Stronger Eye Magnets." By Walter B. Lancaster, M.D., Boston, Mass.

Discussed by Drs. Sweet, Marple, and Lancaster.

14. "A More Exact Method of Measuring Fields of Vision and Arcs of Rotation." By Lucien Howe, M.D., Buffalo, N. Y.

15. "Ocular Phenomena Accompanying Three Cases of Gastro-intestinal Disorder." By Hiram Woods, M.D., Baltimore, Md.

Discussed by Drs. May, Byers, and Woods.

16. "Lumbar Puncture and Examination of the Spinal

Fluid in Affections of the Eye." By James B. Ayer, M.D., Boston, Mass. (By invitation.)

Discussed by Drs. Knapp, de Schweinitz, and Ayer.

17. "Tuberculous Meningitis with Special Reference to the Tubercle of the Choroid and its Pathologic Manifestations." By Frederick Tooke, M.D., Montreal, Can.

Discussed by Drs. Marple, Koller, Randolph, and Derby.

18. "Malignant Uveitis Treated with Thyroid Extract." By James Bordley, Jr., M.D., Baltimore, Md.

Discussed by Drs. Weidler, G. Oram Ring, Randolph, Risley, Rogers, and Bordley.

19. "Progressive Essential Atrophy of All Layers of the Iris." By G. E. de Schweinitz, M.D., Philadelphia.

Discussed by Drs. Zentmayer, Mittendorf, Stieren, Lamb, Risley, and de Schweinitz.

EXECUTIVE SESSION.

Tuesday Evening, July 6th.

The meeting was called to order at 8.30 o'clock, Dr. de Schweinitz presiding.

The Secretary moved that, as the Minutes of the last meeting had been published in the Transactions, the reading of them be dispensed with. (Carried.)

The report of the Council was presented by the Chairman, Dr. Hiram Woods.

The Council recommended the adoption of the Joint Report of the Committees of the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-laryngology Regarding the Establishment of a Joint Board to Arrange, Control, and Supervise Examinations to Test Preparation for Ophthalmic Practice. A copy of this report had been printed and distributed among members of the Society.

The Council presented the following nominations of officers of the Society for the ensuing year:

President: Dr. G. E. de Schweinitz, Philadelphia.

Vice-President: Dr. Peter A. Callan, New York City.

Secretary-Treasurer: Dr. William M. Sweet, Philadelphia.

It was moved and seconded that the Secretary cast a ballot for the nominees presented by the Council, which motion was carried, and the officers were declared elected.

The Council recommended the following named candidates for admission to the Society. No ballot was demanded as provided by the by-laws, and the candidates were therefore declared elected Associate Members.

Dr. Carl Fisher, Rochester, Minn.

Dr. Ellice M. Alger, New York City.

Dr. H. W. Wootton, New York City.

The Council also referred the names of other candidates to the Council for 1916.

The Council in its report urged that, prior to the transmission to the Council of the name of any ophthalmologist applying for membership in the Society, his proposer and seconder should definitely ascertain whether such a candidate possessed the necessary requirements for membership as prescribed by the constitution and by-laws; otherwise it might be found that the candidate's work on his thesis had been expended in vain, because even though the thesis was perfectly acceptable, his name could not be considered by the Council because of technical or other reasons of which his proposer and seconder should have had knowledge.

No recommendation was made by the Council as to the time and place of the next meeting of the Society, as according to custom the Society will meet in Washington, D. C., May 9 and 10, 1916, in connection with the Congress of American Physicians and Surgeons, of which this Society forms a component part.

The Secretary read a communication from Dr. W. R. Steiner, the Secretary of the Congress of American Physicians and Surgeons, asking that a delegate and alternate be appointed to represent the Society on the Committee of Arrangements of the Congress in 1916. Dr. Steiner called attention to the fact that a number of members of the Society who had attended the meeting in 1913 had failed to register as members of the Congress, evidently thinking that registration in the Society was sufficient. The constitution provides that the Secretary is empowered to collect the registration fee of the Congress from members who attend the meeting and fail to register as members of the Congress.

The Chair appointed Drs. L. S. Greene and W. K. Butler as member and alternate on the Committee of Arrangements of the Congress.

The report of the Secretary-Treasurer was presented, and, the Auditing Committee having certified to its correctness, it was accepted and placed on file.

The Secretary announced the death of the following members during the year:

Dr. M. H. Post, St. Louis, Mo.

Dr. E. Gruening, New York City.

Dr. George Strawbridge, Philadelphia.

Dr. Hasket Derby, Boston, Mass.

Dr. J. L. Adams, New York City.

Dr. J. R. McIntosh, St. John, N. B.

The Chair appointed Drs. J. W. Charles, A. E. Ewing, Wilbur B. Marple, S. Lewis Ziegler, and Myles Standish to draft suitable memoirs for publication in the Transactions.

The Report of the Committee on Theses was presented by Dr. Duane:

Dr. Duane: The Thesis Committee had presented to it a number of theses from the applicants. These were passed from one member of the Committee to another and read by all, after which there was a free discussion regarding them,

first by correspondence, and afterward at a meeting of the Committee. Our views were practically unanimous on all points. As this was a new matter, and a precedent had to be established, one thing on which we debated for a long time was what sort of standard should be required in the thesis to be presented; and our feeling was that we should demand a high standard, in conformity with the reputation of this Society. We decided that the thesis should represent original work, original investigation, and a fair examination of the literature of the subject; and should be presented, both as regards form and substance, in a dignified manner, such as would, if the Council deemed right, entitle the paper to be printed in the Transactions and placed in the records of the American Ophthalmological Society. Acting on that opinion, we adopted what may seem to some a rather too critical view; but we felt that at the very beginning it was important to establish a precedent and to determine that the character of the thesis should be such as to be worthy of the Society.

We may say that the theses varied a good deal in merit, but some were of considerable excellence. Evidently most of the applicants took the affair seriously, and prepared theses with great care; and, on the whole, we think this experiment has demonstrated that it is a valuable thing for the Society to have intending applicants present this evidence of their qualifications for admission.

The Committee on Forming a National Organization for the Study of Cancer presented no report, and the Committee was discharged.

The Report of the Delegates to the Clinical Congress of Surgeons of North America was presented by Dr. Stieren:

The Congress was held in London the week before the war. The London physicians entertained the visitors in both a scientific and social way. Clinics were held at the various London hospitals, and the plan of making daily announcements in the assembly room, and issuing cards to the members to visit the clinic desired, worked admirably. The last evening was devoted to ophthalmology, Mr. Walter H. Jessop, presiding, and papers were read by Lt.-Col. Elliott, Mr. F. Richardson Cross, and Mr. J. B. Story.

The Committee on the Standardization of the Illumination of Test Cards and Perimeters, consisting of Drs. Lancaster, Crampton, and Langdon, was unable to complete its work, and the Committee was continued.

The Report of the Committee on Diploma in Ophthalmology had been printed and distributed among the members of the Society, and Dr. G. E. de Schweinitz, Chairman of the Committee, made the following statement:

Dr. de Schweinitz: According to the direction of this Society the Chairman of your Committee on Diploma in Ophthalmology met the Chairmen of similar committees of the Section on Ophthalmology of the American Medical Association and of the American Academy of Ophthalmology and Oto-laryngology. As the result of this conference, a combined report was formulated, and sent to the various members of the three committees and received their endorsement. The report, therefore, is signed by each member of each committee. It has been recommended for adoption by the Council of the American Ophthalmological Society; and a letter received from Dr. Jackson says that the report was unanimously adopted in California at the recent meeting of the Section on Ophthalmology of the American Medical Association. It will also, undoubtedly, be adopted by the Academy of Ophthalmology and Oto-laryngology, which meets in October. It is therefore desirable that this Society, which began this work, shall take a similar action.

It was moved and seconded that the recommendation of the Council, that the Joint Report be adopted, be followed.

Dr. Carmalt asked whether the meaning was that a diploma as a Doctor of Ophthalmology was required before one could become a member of any of these three societies.

Dr. de Schweinitz replied that, other things being equal, the requirement would obtain after 1920.

Dr. Pyle said that it seemed to him that to allow any one to call himself Doctor of Ophthalmology would be contrary to the constitution of this Society.

Dr. May asked for an explanation of the last portion of the report, "The Section on Ophthalmology of the American Medical Association, in so far as it is empowered to adopt its own rules, shall, from the year 1920, require that its officers and those members accorded places on its program shall possess the certificate in question or its equivalent." He wished also to know the meaning of the word "equivalent" in this connection.

Dr. May further said that, unless by equivalent was meant membership in the Society at the present time, a member of the American Ophthalmological Society could not, after 1920, hold an office in the American Medical Association nor read a paper at its meetings.

Dr. Pyle made a motion that the Report be laid on the table. The motion was not seconded.

Dr. Carmalt suggested that the Committee be continued, and thought that it might be possible for them to recommend to the various medical colleges that had a right to confer the degree of Doctor of Medicine that they create a degree of Doctor of Ophthalmology. He did not, however, regard this as a good plan.

Dr. de Schweinitz explained that the matter referred exclusively to those who, in the future, might get degrees, and not to the present members.

Dr. Risley called attention to the fact that everywhere throughout the country there are persons who enter their respective communities and make it known that they are specialists in ophthalmology, although all the preparation they have had, in addition to the regular course in medicine, has probably been a six weeks' course at some polyclinic; and that it had been thought best that something should be done to protect these communities against such men. The Committee thought that the best way to do this was to have these three societies require this special preparation before membership in them could be secured.

Dr. Pyle thought it would be best to defer action until another year, so as to give an opportunity to consider the matter more carefully.

The question of adopting the Joint Report as formulated by the three committees was then put, with the result that the report was adopted.

The report is as follows:

JOINT REPORT OF COMMITTEES OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY, THE SECTION ON OPHTHALMOLOGY OF THE AMERICAN MEDICAL ASSOCIATION, AND THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY REGARDING THE ESTABLISHMENT OF A JOINT BOARD TO ARRANGE, CONTROL, AND SUPERVISE EXAMINATIONS TO TEST PREPARATION FOR OPHTHALMIC PRACTICE.

During the past year the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-laryngology have considered and adopted the reports of their respective committees recommending that graduate courses in ophthalmology representing at least two years of work subsequent to taking the degree of Doctor of Medicine be established in medical schools of the first class; and that such work be recognized by conferring an appropriate degree upon those who have successfully completed it.

It seems clear that there is unanimous agreement as to the need for systematized and standardized training of those who are to practise ophthalmology. At least five of the medical departments of important American universities have now arranged for such courses leading up to special degrees. But already it is clear that the number of graduate students who will take the complete course leading to such a degree will, in the near future, be small. Even in universities capable of furnishing facilities adequate for the training of large numbers the great majority will not meet the requirements for the higher degree.

It is desirable that the standard of attainment for which

university degrees are given should be kept fully up to the present standard of our best universities. As matters now stand, therefore, a large majority of those entering upon the practice of ophthalmology will not be reached, or directly influenced, by these standards. It is extremely desirable that all who take up ophthalmic practice as a specialty should be induced to pursue systematic courses and to show proficiency therein.

Professional opinion, particularly the opinion of those who have already established themselves in ophthalmic practice, can bring about the desired change to a large extent, without assistance from legal enactments and without the necessity for establishing new educational institutions. It is only necessary to bring into existence a competent body to outline the proper course of study to be pursued; to examine and pass judgment upon existing institutions that offer opportunities for such study, and when individual students have proved that they have profited from such opportunities, and have prepared for ophthalmic practice, to give them the advantages of a certificate to that effect.

To accomplish this purpose, in which we believe the ophthalmologists of America are fully united, we make the following recommendations, unanimously agreed to by the joint committees representing the three organizations above named:

1. That, by the conjoint action of the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-laryngology, a Board be established to arrange, control, and supervise examinations, to test the preparation of those who design to enter upon the special or exclusive practice of ophthalmology.

2. That this Board consist of nine members: three to be chosen by each of the above-named organizations, in the same manner as their presiding officers are named. At the first election each organization shall choose three members, one for three years, one for two years, and one for one year; and thereafter one each year to serve for a term of three years. Vacancies shall be filled for the unexpired term by the Society from which the preceding member has been

chosen. No member of the Board shall serve more than six years continuously.

3. Members of the Board shall serve without compensation, but shall be reimbursed for actual expenses while engaged in the work of the Board, provided all other necessary expenses of the Board and its appointees have been properly provided for.

4. The Board shall appoint from its own membership, and from the medical profession outside its membership, a sufficient number of learned and skilled examiners who shall conduct the said examinations and report thereon to the Board.

5. The examinations may be held in any city of the United States where good facilities may be obtained for conducting clinical and practical examinations.

6. The Board shall fix requirements to be met by all candidates for examination, which shall include the successful completion of a course in medicine in a medical school of recognized good standing, at least two years before the examination; adequate study of ophthalmology and allied subjects; and payment of an examination fee to be fixed by the Board. It shall be authorized to prepare lists of medical schools, hospitals, and private instructors recognized as competent to give the required instruction in ophthalmology.

7. Each candidate whom the examiners report as having successfully passed the required examination shall receive by the authority of the Board a certificate or diploma setting forth this fact, but conferring on the recipient no academic degree.

8. The American Ophthalmological Society and the American Academy of Ophthalmology and Oto-laryngology shall from the year 1920 require every candidate for membership in those bodies to possess the certificate above mentioned, unless the applicant shall possess a degree in ophthalmology conferred by a university recognized by them as competent to prepare its students for such a degree. The Section on Ophthalmology of the American Medical Association, in so far as it is empowered to adopt its own rules, shall, from the year 1920, require that its officers and those members accorded places on its program shall possess the certificate in question or its equivalent; and shall request that in the

directory published by the Association the holders of such certificates be especially recognized.

George E. de Schweinitz, Philadelphia.
Myles Standish, Boston.
Samuel D. Risley, Philadelphia.
John E. Weeks, NewYork.
William H. Wilder, Chicago.
Hiram Woods, Baltimore.
Walter R. Parker, Detroit.
Alexander Duane, New York.
William Zentmayer, Philadelphia.
Wendell Reber, Philadelphia.
Walter B. Lancaster, Boston.
Edward Jackson, Denver.

Committee on Diploma in Ophthalmology: In accordance with the provisions of the Joint Report, the Council recommended for election to the Joint Board the following members of the Society: Dr. Myles Standish, to serve for three years; Dr. Alexander Duane, to serve for two years; and Dr. W. H. Wilder, to serve for one year. The Chair announced the election of the members as recommended.

The President announced the appointment of the following committees:

Council: Drs. William H. Wilmer, R. A. Reeve, Alex. Quackenboss, Arnold Knapp, and Wm. Zentmayer.

Committee on Theses: Drs. W. H. Wilder, Alexander Duane, and George S. Derby.

Program Committee: Drs. W. T. Shoemaker, T. B. Holloway, and the Secretary.

Publication Committee: Drs. W. E. Lambert, Robert L. Randolph, and the Secretary.

The Committee appointed to prepare a minute on the death of Dr. M. H. Post presented the following:

It is with deep regret that we are called upon to record the demise of our late associate and President, Dr. M. H. Post. Dr. Post, whose eminence in ophthalmology was

recognized by all who knew of his work, became a member of this Society in 1887. His contributions to the scientific work of the Society although not frequent, were of marked excellence. His social qualities were such as to endear him to all the members of our Society who have had the privilege of knowing him; the recognition of his worth found expression in his election to the presidency of this, the oldest ophthalmological society in America, on its fiftieth anniversary. Not only do the members of the Society recognize a personal loss in the death of Dr. Post, but they feel also that ophthalmological science in America has been deprived of the work of one who has always been faithful to its traditions and in sympathy with its loftiest aspirations.

John E. Weeks
Samuel D. Risley
A. E. Ewing.

Dr. Risley made a motion that a copy of this minute be forwarded to the family of Dr. Post. (Carried.)

MORNING SESSION, JULY 7TH.

The meeting was called to order at 9.10 A. M. by the President, Dr. G. E. de Schweinitz.

The following papers were read and discussed:

20. "The Ophthalmias of Palestine." By Harry Friedenwald, M.D., Baltimore, Md.

Discussed by Drs. Howe, G. Oram Ring, Weeks, and Friedenwald.

21. "Pemphigus of the Conjunctiva. Report of Two Cases." By Edward Stieren, M.D., Pittsburgh, Pa.

Discussed by Drs. Marple, Lambert, and Shumway.

22. "Report of a Case of Colloid Degeneration of the Upper Lid." By Arthur J. Bedell, M.D., Albany, N. Y.

23. "A Case of Epibulbar Sarcoma." By Howard F. Hansell, M.D., Philadelphia, Pa. (Read by title.)

24. "Multiple Melanotic Nevi of the Conjunctiva, with Malignant Invasion of the Corneal Limbus." By F. P. Capron, M.D., Providence, R. I.

Discussed by Dr. de Schweinitz.

25. "Report of Two Cases of Metastatic Carcinoma of the Choroid and One Case of Myxosarcoma of the Orbit." By John E. Weeks, M.D., New York City.

Discussed by Drs. May and Maxson.

26. "A Case of Primary Intradural Tumor of the Optic Nerve." By Charles R. Heed, M.D., Philadelphia.

27. "A Primary Intradural Tumor of the Optic Nerve Successfully Removed, with Preservation of the Eyeball, by the Krönlein Method." By Arnold Knapp, M.D., New York City.

28. "A Contribution to the Subject of Tumors of the Eyelid and Orbit." By G. E. de Schweinitz, M.D., Philadelphia.

The papers of Drs. Knapp, Heed, and de Schweinitz were discussed by Drs. Carmalt and de Schweinitz.

29. "Hereditary Posterior Polar Cataract, with Report of a Pedigree." By S. Lewis Ziegler, M.D., and J. Milton Griscom, M.D., Philadelphia.

Discussed by Dr. Ziegler.

30. "Family Degeneration of the Macula Lutea, with a Suggestion as to its Cause." By Brown Pusey, M.D., Chicago, Ill.

Discussed by Drs. de Schweinitz, Weeks, Wilder, and Pusey.

31. "Retinal Disease with Massive Exudation and an Allied Condition. Report of Two Cases." By William Zentmayer, M.D., Philadelphia.

Discussed by Drs. G. Oram Ring and Mittendorf.

AFTERNOON SESSION, JULY 7TH.

The meeting was called to order at 2.10 P. M. by the President.

The following papers were read and discussed.

32. "Spontaneous Reattachment of the Retina after Twenty-two Years' Duration. Report of a Case." By A. Edward Davis, M.D., New York City.

Discussed by Drs. Sutphen, Bedell, Byers, Lambert, Ziegler, and Davis.

33. "Anomalies of the Accommodation Clinically Considered." By Alexander Duane, M.D., New York City.

Discussed by Drs. Ziegler, Howe, Zentmayer, Davis, de Schweinitz, Holden, and Duane.

34. "Nervous Symptoms from Contusions of the Eye." By Edward R. Williams, M.D., Boston, Mass.

Discussed by Drs. Derby and Maxson.

Adjourned.

Members present at the Fifty-first Annual Meeting:

Alling, Arthur N.
Bailey, Fred. D.
Bedell, A. J.
Bordley, James, Jr.
Buckman, E. U.
Butler, W. K.
Byers, W. G. M.
Callan, Lewis White
Callan, Peter A.
Capron, F. P.
Carmalt, W. H.
Chandler, Henry B.
Claiborne, J. Herbert
Clap, Edmund W.
Cutler, Colman W.
Davis, A. E.
Derby, George S.
Duane, Alexander
Duncan, J. L.
Ewing, A. E.
Foster, Matthias L.
Fox, William H.
Francis, Lee Masten
Fridenberg, Percy
Friedenwald, Harry
Goldberg, Harold G.
Greene, Louis S.
Griscom, J. Milton
Hansell, Howard F.
Harrower, David
Haskell, Henry H.
Heckel, Edward B.
Heed, C. R.
Hepburn, N. J.
Holden, Ward A.
Holloway, T. B.
Howe, Lucien
Hunter, D. W.
Ingalls, James W.
Jack, Edwin E.
Jameson, P. Chalmers
Kilburn, Henry W.
Knapp, Arnold
Koller, Carl

Lamb, Robert Scott
Lambert, Walter Eyre
Lancaster, Walter B.
Langdon, H. Maxwell
Lovell, D. B.
Lowell, W. Holbrook
Macleish, A. L.
Marlow, F. W.
Marple, W. B.
Maxson, S. C.
May, Charles H.
Miles, Henry S.
Mittendorf, W. F.
Pooley, Thos. R.
Posey, W. C.
Pusey, Brown
Pyle, Walter L.
Radcliffe, McCluney
Randolph, R. L.
Reeve, R. A.
Reynolds, W. G.
Ring, G. Oram
Ring, Henry W.
Risley, Samuel D.
Rogers, W. K.
Sattler, Robert
Sautter, A. C.
Schweinitz, G. E. de
Schwenk, Peter N. K.
Shannon, John R.
Shoemaker, J. F.
Shoemaker, Wm. T.
Shumway, Edward A.
Smith, E. Terry
Snell, Albert C.
Standish, Myles
Stieren, Edward
Sutphen, T. Y.
Sweet, W. M.
Thomson, Edgar S.
Tooke, Fred.
Tyson, H. H.
Verhoeff, F. H.
Waite, Frank L.
Weeks, John E.
Weidler, Walter Baer
Wescott, Cassius D.
Wilder, Wm. H.
Williams, Carl
Williams, Chas. H.
Williams, Edw. R.
Wilmer, W. H.
Wilson, F. M.
Wood, J. Scott
Woods, Hiram
Zentmayer, W.
Ziegler, S. Lewis

A REVIEW OF THE GLAUCOMA QUESTION, WITH SPECIAL REFERENCE TO NEWER CONCLUSIONS OF ITS ETIOLOGY AND DEPARTURES OF ITS SURGICAL TREATMENT.

ROBERT SATTLER, M.D.,
Cincinnati, Ohio.

The present paper considers conclusions, that the origin of acute primary glaucoma may be largely or wholly dependent upon an invalidation or loss of ocular dynamics, and that, like the dynamics of the body as a whole, correlated and balanced kinetic activities are spontaneously initiated and conserved along uniform lines during consecutive age periods, from infancy to senility, through energic impulses furnished by the central and sympathetic nervous systems, and these interrupted or lost, if congenital or subsequent accidents or anatomic anomalies, encroachments of senescence or pathologic invasions, force their temporary or permanent suspension.

That each smaller differentiated whole or integral part of our human economy, whether as organs of greater or lesser functional importance or only as territorially differentiated parts, have their own kinetic systems which preside over and maintain a pressure equilibrium securing unimpaired protection, nutrition, and respective function of the specialized cells of the organs or territories in question.

That with the more complex differentiation of tissue and specific function of higher mental, brain, and special sense neurons we note what may be considered distinctive anatomic characters marking their respective kinetic systems, and thereby conserving and making less vulnerable their inherent higher physiologic purposes.

That, furthermore, the facts mentioned may furnish us a more rational explanation of kindred lesions of the internal ear and brain, which in the mystery of their origin resemble glaucoma, in the diagnosis of which we assume or actually prove the presence also of the distinctive physical sign of excessive tension; that one or all may have a probable common origin, in a disturbance of their own or contiguously related kinetic systems.

The foremost disclosure which a study of the human eyeball during consecutive age periods affords us is that we must keep in mind that a relatively considered normal globe tension or globe resiliency represents a more or less perfect equilibrium between two opposing and correlated kinetic activities, within the limit of which adequate nutrition and undisturbed function of the specific visual percept neurons of the retina become possible and permanent physical and physiologic facts. In common with those of all vertebrates, the human globe has a strong capsule tunic for the safe and concealed lodgment and protection of its visual neurons. Through anatomic balance of its (motive) circulatory force and the inherent elastic (static) one of its strong tunic wall, not only is the shape of a spheroidal camera obscura conserved, but unimpaired functional activity or vision percepts are made possible for life. It is probable that slight and passing degrees of greater than a relatively accepted globe hardness or softness, but not permanently harmful to function, happen constantly and frequently in normal eyes of otherwise normal individuals; from manifold emotional causes or their circulatory counterparts. These are promptly and spontaneously adjusted through the interaction of the inherent kinetic attributes of the circulatory channels and of the tissue-resiliency of the capsular tunic which, in addition, is reinforced by regional safeguards with the quality of latent and greater compensating elasticity, *i. e.*, the lamina cribrosa of the sclerotic, the cornea, the entrance and the exit openings

of the short and long ciliary arteries and veins, the pectinate ligament anteriorly, and the posterior choroidal ring, and between these the vortex scleral openings for the passage of the larger emissary veins.

If undiscoverable congenital anatomic variations of the tunic capsule or similar anomalies of the circulatory channels are present, spontaneous recovery of a stable equilibrium of the forces concerned is more difficult, and, we can assume, is mainly brought about through a more persistent and responsive call upon the regional safeguards referred to. Also, that the, at times, greater subjective discomfort and disordered visual percepts which are common to individuals with eyes burdened with such congenital or subsequently acquired circulatory hindrances, or an anomalous capsule tunic, are inordinately increased during emotional and mental excitement or depression, and that transitory blood toxicity from various causes, vascular encroachments of senescence, also disturb and influence recovery of a more uniform pressure balance. We have also many opportunities to observe that definite pathologic intraocular causes, accompanied in some cases by intangible and in others by discoverable evidences of chemical or biochemical changes in the composition of the aqueous and vitreous contents, afford us the earliest proof of a shorter or longer lasting impairment of general or regional globe elasticity, with coincident stasis and prolonged retention of the intraocular blood and lymph volume.

In other words, we note the incipiency of that which may terminate in invalidation, followed by more or less lasting interruption of one or the other, and, finally, in a loss of ocular kinetics which essentially govern and conserve unimpaired function of the specific visual and ganglion-cells of the retina and those of the mixed and sympathetic ganglia of the uveal tract. Fetal existence also furnishes us convincing proof that a preponderant loss or increase of one or the other

of the motive forces concerned in the conservation of globe tension, commensurate with a normal progressive general and ocular development, is not an infrequent happening, if hereditary impulses or anatomic deflections have added impediments to an otherwise normal intraocular circulatory system, or intra-uterine pathologic changes have burdened or exhausted the similar motive force of the capsule tunic, or *vice versa*. In such cases we have at birth a hard, enlarged eye with clinical and pathologic evidence of congenital primary glaucoma or the soft shrunken globe of a similarly destructive sclerokeratitis or iridochoroiditis. The assumption is a reasonable one that, even during embryonic life, lasting or even fatal dangers which, in consequence of more prolonged excessive or reduced tension, would prove harmful to the retinal neurons, are repeatedly and frequently averted by spontaneous adjustments in utero, as the fetal globe may be inherently endowed with the same or even a greater quality of compensatory general and regional elasticity and overcomes as promptly the transitory or more lasting blood and lymph stasis and its coincidental defective metabolism by repeated recovery of normal globe tension as it is known to take place during earlier and later life periods.

The physical sign of transitory or permanent globe hardness or softness, above or below a relatively accepted and tentatively standardized tension, and made evident through an educated touch and the perfected precision of tonometers, marks every interruption or loss of ocular kinetics.

The probable explanation of this happening is that a retarded or quickened intraocular blood and lymph volume with possible but only slight volumetric increase or decrease must, unless followed by prompt recovery of a more stable or normal equilibrium or tension, be attended by next to simultaneous destructive metabolic physicochemical changes of the retina and its neurons, as well as of the colloid structures.

Whether attended by the physical sign of high or low tension, the transition from one to the other, or the more permanent presence of one only, marks a disturbance or a complete forfeiture of globe kinetics.

The circulatory forces which so abundantly furnish blood and lymph to the intraocular structures have distinctive anatomic characteristics. The main blood and lymph reservoir, the uveal tract, has arterial and venous channels which, in their terminal ramifications, are more independent of one another.

The delivery of arterial blood once accomplished over limited but wider territorial areas, through a most intricate system of larger and smaller capillaries, prevents its longer retention or volumetric increase; an even finer meshwork of venous radicals, which at the same time serves as a supply and drain for the huge lymph lakes which surround and permeate the dioptric media, and of which they are besides an integral part, together with the large emissary or vorticose veins, accomplish a similar unimpeded outflow of lymph and venous blood.

It is not an improbable assumption that the close resemblance of the vascular systems and independent or restricted territorial distribution of the blood-vessels of the choroid to the similar ones of certain brain areas and of the internal ear, may explain the dynamic disturbances which, in a modified form, are common to one and all, and that the physical sign of an excessive decrease or increase of intraocular, intracerebral, and intralabyrinthine pressure may be due to the same or similar causes which disturb or exhaust opposing but correlated motive forces which maintain a stable pressure, consistent with adequate nutrition and undisturbed function of the brain, retinal and internal ear neurons.

The circulation of the retina, a single or exclusive artery furnishing blood plasma to its principal ganglion group, its neuro-epithelial group receiving a similar exclusive blood

volume from the choroidal circulation, are even more striking analogous comparisons that certain restricted or apportioned brain areas harboring neurons for the higher brain and mental functions resemble in their kinetic attributes the same or similar ones of the eye. This may explain what is abundantly upheld by corroborated facts, that pronounced cardiovascular lesions and general or wide-spread arteriosclerosis, chronic nephritis or hepatic cirrhosis, uremia, the last stages of diabetes or lesions of the ductless glands attended by high or low blood pressure, rarely influence harmfully, and exceptionally only if preëxisting or coincident intraocular causes are active also, the motive forces concerned with the conservation of a relatively normal intraocular tension. To this we may add that, even in those cases of consecutive or independent ocular arteriosclerosis of the central artery of the retina and its vein, made strikingly evident to us through ophthalmoscopic disclosures, even cyanosis and ischemia, thrombus or embolus, stasis, contraction or corrugation of the retinal vessels from local and visible endarteritic or perivascular changes, rarely attend or cause interruption of ocular kinetics or influence unfavorably the relatively normal intraocular tension.

It is even more widely known and accepted that blood toxicity from many and various causes, to mention only neuronal disintegration of focal and other brain lesions, grave hemic disorders, etc., even though the integrity of the retinal neurons may be impaired or destroyed, globe kinetics, *i. e.*, intraocular tension, is neither influenced nor impaired. Nor is it markedly influenced and rarely upset by even the most pronounced focal ocular lesions or widely disseminated pathologic processes of the uvea, retina, or sclerocorneal tunics in which the major blood or lymph volume is not completely shut off from within or without; exceptions to this are offered us by traumatic and pathologic lesions which temporarily bring about a sudden stoppage to the ingress of

all arterial blood or prevent as suddenly the exit of *all* lymph and venous blood, *i. e.*, excessive swelling of traumatic cataract, intraocular tumors which have exceeded their displacement, incipient stage of panophthalmitis, septic and metastatic choroiditis, etc.

The interruption or loss of ocular kinetics, with the physical sign of reduced or lost globe tension, suggests as probable that a more or less rapid and greatly minimized, slow, or lasting shutting off of the entire volume of arterial blood, which the eye receives through the long and short ciliary arteries, has resulted.

Apt clinical and pathologic illustration of this we have in sympathetic uveitis and in cases of malignant (idiopathic) uveitis, and in rare expressions of sclerokeratitis of adolescence, often erroneously attributed to congenital lues, but with greater probability assignable to tuberculosis.

Interruption or loss of ocular kinetics with the physical sign of moderate or excessive globe hardness is probably dependent upon a sudden transitory or more lasting complete strangulation or impoverishment of the entire intraocular volume of lymph and venous blood.

Hypertonus is the cardinal clinical sign of acute glaucoma and its subsequent phases, whereas hypotonus is equally characteristic of sympathetic and other varieties of uveitis. Hypertonus, or abnormally increased globe tension, strongly suggests that a longer retention or imprisonment of the entire intraocular blood and lymph volume, containing effete toxic and other physicochemical and even gaseous products of neuronal degeneration, is about to or has taken place, and hypotonus, or abnormally reduced globe tension, is consequent upon and coincident with a slower and lasting withdrawal of the entire supply of arterial blood, with its voluminous share of oxygen-bearing leukocytes.

It furthermore upholds that it is not the excessive increase of intraocular pressure, largely a physicochemical consequence, which causes undue or destructive intraretinal com-

pression or separation of the multitudinous perikaryones, their axones and dendrones with invalidation of their synapses, but that it is a necessary result of an antecedent and fleet induction of a degeneration of retinal neurons and the colloid structures from want of oxygen and preponderant and cumulative presence of imprisoned effete and toxic intraocular products.

If we accept the evidence at hand that the neurons of the retina represent a projected unit of innumerable similar ones of the brain subserving higher and lower mental and brain functions, it becomes patent at once that brain and ocular dynamics have much in common. The apparent difference is that the former (brain) represents a multiple union of similar kinetic systems which preside over and conserve a stable relationship of secure protection and adequate nutrition for unimpaired function of many independent or particular groups and of numerous other anatomically and physiologically connected groups of neurons, whereas the latter (eye) has a single kinetic system only for the unharmed preservation and function of its visual percept neurons. Any embarrassment of its own or a similarly exclusive but allied contiguous intermediate or central brain kinetic system in one or several divisions of the visual path fibers, from percept (retina) to primary optic centers, thence (optic thalamus, corpus geniculatus and anterior corpus quadrigemina) to the cortical half vision center in the anterior part of the occipital cortex divided by the calcarine fissure into an upper or cuneate and a lower or lingual lobe, and thence to the great association centers of the cerebral cortex, will briefly or permanently interrupt the entire continuity of transmission of their respective and particularized parts and pervert or prevent light or color perception or the still more complex psychic process of vision. We may at once interpose the statement that it is not because of the present higher or lower intracranial pressure of a focal or diffuse hidden brain

lesion, for with even greater probability we can assume that it is due to a direct or first sequence of the upset of its own or a related contiguous kinetic system, and the neuronal degeneration it induces or causes in one or all of its component parts.

This statement stands or falls unless we can prove by scientific facts that the analogy between the motive forces of the brain and eye, brain and ear, and other percept outposts or projecting brain neurons of special sense are next to identical in kind and purpose. In other words, that an excessively high or low, assumed or actual, intracranial, ocular, or vestibulolabyrinthine tension, accompanying certain pathologic changes, is for one and all a reliable and sufficiently constant physical sign which predicates an imminent or already active degeneration or destructive metabolism of a group of functionally related groups of neurons, hitherto subserving unimpaired specific brain and mental function. Furthermore, that their special function of visual percept and sensation was alone accomplished, prior to pathologic invasion, through the interaction of allied and balanced motive forces, congenitally present and active since birth, which neither heredity nor accident having hindered, will conserve for life through a more or less circumscribed neuroglia envelop, static force, or territorial grouping and a correlated more variable motive circulatory one, their inherent special functional activities.

The human globe, harboring a single group of functionally highly differentiated brain retinal neurons and their complement of sympathetic ganglia, is additionally reënforced by the bony enclosure of the orbit; the brain, protecting and conserving the specific functions of many independent and correlated groups of similar neurons and their larger complements of sympathetic ganglia, is surrounded or encased by an even more compact and unyielding bony cranium. Neither orbit nor skull is essential to single retinal or complex brain

and mental functions, but are only additional safeguards against accidents to eye and brain from without and within. It is even probable that, in the light of present-day facts, the capsule tunic of the eye and the dural envelop of the brain contribute almost exclusively to the lasting security and protection of their respective neurons, and that neither permits more than a fractional degree of greater compressibility or separation of their synapses, dendrones, or axones above or below a uniform pressure equilibrium which neither interferes with nor shuts off the approach of a plentiful supply of blood plasma or oxygen-bearing leukocytes which the finest ramifications of capillaries deliver to their allotted brain or retinal regions. It furnishes convincing proof of two additional facts: the first that, for both eye and brain, the physical sign of increased tension does not precede but follows an antecedent hidden disturbance of a single (eye) kinetic system or one or several (brain) correlated multiple ones, connecting and conserving the territorial integrity of contiguous particularized aggregations of brain neurons; the second, that, for both, an opening of the capsule tunic of the eye and the dural envelop of the brain effects prompt recovery of a transitory or lost stable pressure equilibrium marked by the lessening or disappearance of the physical sign of excessive tension. A carefully planned and executed limited incision through the entire thickness of the capsule tunic at or within the sclerocorneal margin, topographically determined as the safest by the cumulative experience of generations of ophthalmic surgeons, accomplishes a restoration of ocular dynamics, and with equal certainty can relief, from a hidden antecedent pathologic disturbance of the brain, general or localized, be counted upon to follow an opening of the dura which has resulted in a partial or more complete upsetting of one or several contiguously related kinetic systems of the brain. Its minimum or maximum degree of abnormal pressure, however, is not caused, as commonly held, by the

intact and unyielding wall of the skull, but with greater probability is assignable to the narrow limitation which the dura itself imposes to even minor degrees of volume increase harmful to the brain neurons, like the capsule tunic of the eye, for its retinal group of neurons. To relieve the pent-up, single or several, brain kinetic systems not only must the skull be opened, but, far more essential, an incision or free opening of the dural envelop is prerequisite before relief can or will take place. The placing or exact topography for this opening incision of the dura may comprise a newer question and become the dominant one of the intricate subject of focal brain localization. A more careful study of brain dynamics, as similar conclusions have proved for the eye, may direct a more conservative and beneficent surgery, if not for cortical and subcortical tumors, certainly for some congenital cerebellar cysts of slower growth, and the cystic and solid neoplasms of the hypophysial region of interminably slow growth and, for this reason, of acknowledged benign character. For both, as is known, present major brain surgery involves enormous risk to brain and life, owing to the difficulty of access to these hidden localities through the trans-sphenoidal route or frontal osteoplastic methods.

In truth we have no tangible clinical and scientific proof that slow or sudden, slight or excessive, increase of a relatively considered normal globe tension is, as has been so long tacitly accepted, the cause for acute glaucoma, nor can we assume a similar conclusion that low tension, variable in degree, but not in kind, is the cause of sympathetic or malignant uveitis; it is far more reasonable to assume that both are only opposite manifestations of an impaired or lost globe kinetics of which higher or lower than normal tension is only the physical sign marking an earlier or later stage of progress of an antecedent hidden intraocular destructive metabolism which is the real or primary cause. A greater or lesser degree of globe hardness in acute primary glaucoma,

and of its unerringly diagnosed later phases, can only be regarded as a more certain physical sign that a concealed or hidden degeneration of the retinal neurons is or has been in progress, and indefinitely only does it mark the stage and progress of an antecedent lesion which endangers the, until then, spontaneously initiated and conserved kinetics of the eye. The immediate cause for the presence or absence of this physical sign of excessive globe tension is that it signals a variable degree of a longer lasting imprisonment of a maximum intraocular blood and lymph volume, presumably deprived of its oxygen and loaded with other effete and toxic products of the neuronal, colloid, and other tissue waste.

It would appear that the ostensible aim of the many newer surgical measures, which the inventive ingenuity of earnest investigators has proposed and practised through multiferous schemes, theories, and modifications of new and old surgical methods, is mainly directed against the much battered target of high tension. Efforts to prevent the appearance of the physical sign of higher tension through greater precision in diagnosis during the imminent stage, and to more speedily practise a commonly and almost universally approved surgery, at the incidence of true primary glaucomatous lesions, would often have served a more rational purpose.

This is the reason also that the newer surgical methods,—trephining of the globe, cyclodialysis, iridotasis, etc.,—as well as the longer tried and accepted ones,—iridectomy, sclerotomy, etc., which direct their aim solely to the reduction of high tension in true and so-called glaucoma, with this purpose alone in view,—must often fail or even prove harmful. It is a next to indisputable fact that acute glaucoma and its earlier prodromata and clinical phases are, for a preponderant percentage of cases, curable through a commonly accepted, carefully planned, and successfully executed surgical procedure. In addition cumulative surgical ex-

perience unequivocally selects and holds to an exact topography for the initial incision or opening of the globe. That it must be at the limbus or within the sclerocorneal margin has never been questioned, for it secures the greatest safety of technical execution and beneficent returns to the patient if diagnostic ability and surgical proficiency combine to bring this about. All former and recent surgical departures from old or newly invented methods do not assail the location for the initial opening of the globe, but mainly its breadth and linear character. Whether the round opening of the trephine or the linear one of the knife will uphold its respective claim for greater safety, facility of execution, modern conclusion leaves an open question, to be determined by future individual and common surgical experience.

Present-day conclusions of acute primary glaucoma permit us only to uphold, with increasing probability of confirmation, that the physical sign of high tension, even before it is conspicuously present to touch or tonometer, indicates an unmistakable, often from the first fatal, threat to the retinal neurons, and that it more frequently follows than is coincident with a destructive, pent-up intraocular toxicity: that for the majority of cases of true acute glaucoma and its subsequent phases anatomic hindrances of the capsule tunic or circulatory forces, coupled inseparably with psychopathic tendencies, precipitate their clinical declaration.

That the beneficial returns of opportune and successful surgery in the main effect a temporary or lasting restoration of balance of power to the capsule tunic by establishing an artificial safeguard at the most favorable location, with compensating elastic property, similar in purpose to the natural or inherent ones, which have been exhausted or lost.

DISCUSSION.

DR. CARL KOLLER, New York: While I agree with Dr. Sattler in many respects, I wish to take exception to one

of the sentences in the abstract of his paper, which reads: "The newer and long accepted surgical measures, which direct their aim solely at the reduction of high tension in true and so-called glaucoma, must not only often fail, but even prove harmful." High tension is the most prominent symptom and, so to say, the very essence of glaucoma, even though the pathologic processes leading to it are manifold. There is no doubt about the desirability and reasonableness of treating the causal conditions radically, not stopping at removing the one symptom of increased intraocular tension. But the lowering of the tension is our most urgent task, and frequently the only means of saving the threatened loss or deterioration of vision. For fifty or sixty years, long before much attention was paid to the general conditions underlying it, we have learned from Albrecht von Graefe how to cure inflammatory glaucoma by performing an iridectomy, and innumerable eyes have been saved by this procedure alone. Increasing knowledge and insight will, no doubt, lead to prevention and cure of the underlying causes, but until this is achieved we will have, at the same time, to concentrate our attention upon the operative cure of increased tension. The surgery of glaucoma stands in the present decade in the foreground of interest, and, as I have made a long study of it, but never published anything about it, I take this opportunity to give my experience.

I shall speak first of inflammatory glaucoma. Almost every acute glaucoma can be reduced before operation, and should be reduced before attempting operation. By this the operation is made easy and deprived of its dangers, the wounding of the lens, prolapse of iris, inclosure of iris angles, hemorrhages after operation, and sometimes acute glaucomatous attacks after operation.

The reduction of glaucoma is accomplished partly by general means, indirectly stimulating the heart and circulation, partly by local means. At first a purge is given to the patient, which removes a certain amount of toxins, and then bromid internally and morphin subcutaneously are liberally administered. The local means applied are alternate instillation of eserin (alkaloid) in ½ per cent. *neutral* olive oil, and dionin, 10 per cent. once every hour until the pupil is narrow. Afterwards pilocarpin muriate, 2 per cent., is

instilled every two hours up to the time of the operation. After the reduction the necessity for general anesthesia at the operation is entirely eliminated.

The patient in the days before the operation is trained several times by introducing the speculum, and exercised in the movements of the eye, while the strong electric illumination is turned on, as at the operation. Two and one-half hours before the operation $\frac{1}{6}$ grain morphin is injected hypodermically, and one-half hour before the operation again $\frac{1}{6}$ or $\frac{1}{4}$ grain is given. Cocain, 5 per cent., is injected subconjunctivally, nasally, below, and temporally, but *not* at the site of the incision above. As a preliminary to every glaucoma iridectomy, posterior sclerotomy is performed with the Graefe knife. The operator will notice that in most cases the liquid escaping is water, and not vitreous, a fact pointed out first and emphasized by Dr. Arnold Knapp. This sheds a good deal of light on the pathology, with which I cannot deal now. As a consequence of this sclerotomy the lens at once sinks back and the anterior chamber becomes deep. The iridectomy following is very easy in the now softened eye, only one has to employ an especially sharp keratome. There is no need of replacing the angles of the iris, as the iris will spring back to its normal position, and there is no danger of inclosure. The healing is prompt. I have never seen any harm from the posterior sclerotomy, and never observed intraocular hemorrhage. As its only trace, one can see later with the ophthalmoscope a diamond-shaped white area of choroidal atrophy at the site of the incision.

DR. W. B. MARPLE, New York: I would say that I have not been so successful, as Dr. Koller evidently has been, in reducing the tension by preliminary treatment. All my cases of acute glaucoma are put to bed at once and purged. Then morphin is given, followed by instillations of a solution containing dionin, 5 per cent., eserin, ½ per cent., and pilocarpin, 2 per cent. I often succeed in reducing the tension, so far as the tonometer is concerned, say from 80 to 65, or possibly lower; but, as regards any considerable effect in contracting the pupil and increasing the depth of the anterior chamber, so as to facilitate the performance of an operation, my success has not been so great as Dr. Koller states

his to have been, viz., reducing the tension in nine out of ten of the acute cases. If I do so in four or five, or even less, out of ten, I am satisfied.

DR. JOHN E. WEEKS, New York: I should like to say a word regarding the subconjunctival injection of cocain. I have obtained, in some of my cases, a systemic reaction that has been somewhat distressing to the patient, although not alarming to the operator. A 2 per cent. solution is sufficient in most cases. I use it almost invariably before the operation in cases of glaucoma, injecting 2 to 4 minims in the horizontal meridian on each side of the cornea. The effect in reducing the sensibility of the iris is very marked. I wish to express my indebtedness to Dr. Koller for the suggestion of using it which he made years ago. I have been employing it in this way for nine or ten years.

DR. COLMAN W. CUTLER, New York: I should like to ask whether dionin is always considered a safe drug to use in acute glaucoma. There have been cases reported in which the tension has been said to have risen after the use of dionin, and I think that I have had such an experience; but it is difficult to say whether the tension would not have risen without its use. The theory, I presume, would be that the transudate blocks the channel through which the intraocular fluid must escape. I should like to ask what has been the experience of the members of the Society in regard to this.

DR. WILLIAM H. WILMER, Washington, D. C.: I should like to ask the author of the paper whether, in his experience, he has found that he is able, in chronic glaucoma, to do away with operative procedures. Glaucoma is a complicated process, and we know very little about it. While we are studying the biochemical changes, is it not wise to operate and save the eye? Our duty is to save the patient's eye, and not merely to get a scientific explanation of the biochemical changes going on. We should, of course, study these changes; but, when we have safe measures of operative procedure, we should operate. Sometimes, when we know that there is intestinal putrefaction going on, we can do good by giving subconjunctival injections of common

salt solution, sodium bicarbonate, etc. If, however, these methods fail, are we to let the eye go? We must do everything that we can to save the eye. We know the methods by which we can get the best results from operation. Some prefer one operation, and some another, but it is our duty to operate, while continuing our scientific investigations of the causes of the changes that occur in glaucoma.

DR. ROBERT SATTLER, Cincinnati: The concluding sentences of the paper may convey a partial answer to Drs. Koller and Wilmer: First, that in acute primary glaucoma the eyeball must be opened as speedily as possible to relieve an imminent or actually present blood and lymph stasis, and consequent intraocular toxicity, which is certain, unless promptly averted, to jeopardize or even to destroy the retinal neurons. That it is not the excessive tension or globe hardness which is the cause of acute glaucoma or its later phases, but, with greater probability, a disturbance of ocular dynamics which precipitates, first, a neuronal degeneration of the retina and coincidentally also similar biochemical ones of the colloid structures, with transitory or permanent suspension of function; and that the physical sign of excessive intraocular tension more often follows than precedes the latter. That the points in question are not the location or exact topography for the incision, for it is universally conceded that the sclerocorneal limbus is the safest regional one for the initial opening of the globe, but that the contentions of surgical departures mainly concern or center around its character, whether a free linear one, made with a knife, or a circular one, with a trephine, be selected or preferred.

Like Dr. Koller, it was with me formerly a routine treatment first to resort to the systematic and energetic use of miotics and other local and constitutional measures during the earlier phases of acute glaucoma, and subsequently practise or advise surgical treatment (iridectomy). In recent years, convinced of the presence of physical causes interfering with or upsetting the dynamics of the eyeball, and also that an abnormal psychology is commonly present or can with rare exceptions be discovered, insistence upon the advisability of more prompt surgical treatment has, in latter years, been

an invariable practice, even during the incidence or prodromatic and earlier clinical phases of acute glaucoma.

In reference to the choice of a trephine or linear knife-opening of the globe for the relief of acute primary glaucoma, my personal experience upholds unreservedly the linear-opening incision; for more advanced phases of acute and chronic glaucoma, with the dominant physical sign of excessive tension and serious invalidations of function, I am not at present prepared to add my experience preponderantly for or against one or the other.

A REPORT OF SEVERAL CASES OF CHRONIC SIMPLE GLAUCOMA TREATED BY IRIDOTASIS.

DAVID HARROWER, M.D.,
Worcester, Mass.

Two years ago, at Washington, I reported two cases of iridotasis, of which the results were very satisfactory and have so remained. Indeed, they were so good that I have done only this operation for chronic glaucoma since. The simplicity, the freedom from irritation following, the rapid recovery, and the permanent results so far are all strong arguments in its favor.

I will briefly describe the operation. Half an hour before operating a 1 per cent. solution of atropin sulphate is instilled. I use a 4 per cent. solution of cocain, dropping it in the eye at intervals of two minutes for six minutes. After the speculum is introduced, I ask the patient to look down, and grasp the conjunctiva about 10 mm. above the sclero-corneal margin with the fixation forceps. I make a cut about 6 or 8 mm. long, transversely, going well down to the sclera, and dissecting the conjunctiva closely to the sclera to the corneal margin.

If the patient is quiet, I use no other fixation than the

one I have described. I am very careful not to perforate the conjunctiva, as this would be likely to expose the iris to infection. (If the patient is nervous, I have an assistant fix the eye for me below.) Then, using a keratome, I enter the sclera just behind the cornea, making a wound about 4 to 5 mm. I then introduce the iris forceps, and grasping the iris at the free edge, pull it into the wound, leaving the under surface of the iris up. The conjunctiva is pulled back in place, and the eyelid raised, and then closed. The patient is up the next day, and generally can leave the hospital in four or five days. There is no pain; there is no discomfort.

The cases I reported two years ago are both in excellent condition. The following cases have been operated on since that time:

CASE 1.—Mrs. A. K. P. Referred by Dr. Harriman, of Whitinsville. In July, 1911, V. R. E. = $\frac{10}{10}$, field normal; L. E. = $\frac{4}{10}$, nasal field limited. Pilocarpin was ordered, and in August the field had improved. V. L. E. = $\frac{4}{10}$. In November, V. L. E. = $\frac{5}{10}$ + .75 D. cyl. axis 180 = $\frac{8}{10}$. I did not see her again until February 10, 1912, when her vision was only $\frac{3}{10}$. Two weeks later the vision under pilocarpin was $\frac{4}{10}$.

I did not see her again until May 10, 1913. She had been ill with sciatica, and had had an operation of stretching the nerve. V. = fingers at eight feet. I tried pilocarpin with no results. I did an iridotasis May 17th. One month later the vision was $\frac{2}{10}$, at which it has remained.

The field (Fig. 1) was taken May 10th, the week before the operation. This is shown by the dotted lines. The continuous line shows the field taken July 11th, two months later. The right eye has been controlled by pilocarpin, and the field and tension are normal.

CASE 2.—Mr. P. L. Examination, March 11, 1915. Patient stated that eight months previously his vision had failed completely. For the last two weeks he has had considerable pain in the right eye. The eye was hard, tension

being 57 mm. The pupil was much dilated. I operated the next morning under cocain. When I attempted to draw out the iris it broke off, and only pigment came out with the iris forceps. I tried several times with the same result. I then bandaged the eye, and sent the patient back to the ward. For several days he had relief from his pain; then the eye began to pain him again, and he had increased tension. My assistant, Dr. O'Connor, did a paracentesis, and this relieved him until March 23d, when his eye began

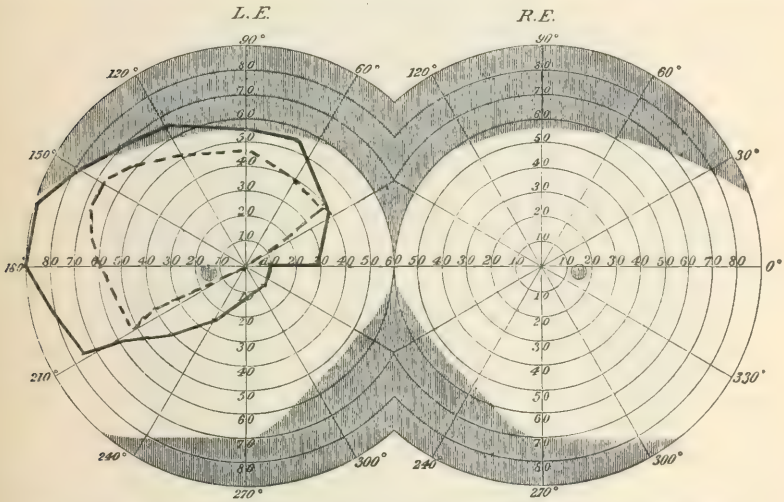


Fig. 1.—Dotted line, field before operation; solid line, after operation.

to pain him again. I then tried an iridotasis down, and was entirely successful. The eye cleared up in a few days, and I sent him home. I have seen him only three or four times since then, and he has been entirely free from any pain. His eye is clear, and he is able to pursue his business as a gardener without any inconvenience.

CASE 3.—Mrs. E. P. C., aged seventy. Examination, April 3, 1914. Has had inflamed eyes for six weeks, starting with the right eye. Has been using atropin, two grains to the ounce. Sees rainbow colors.

V. R. E. = $\frac{4}{200}$; L. E. = $\frac{8}{200}$. Pupils widely dilated.

I did not use the tonometer, as the tension was R. E. + 1; L. E. + 2.

I did an iridotasis on the right eye at once, and the following day on the left eye.

Patient did well, and went home within a week. I saw her in my office April 15th. Tension normal in each eye. R. E. V. = $\frac{1}{10}$, + 1.50 D. cyl. axis 30° = $\frac{4}{10}$; L. E. V. = $\frac{3}{10}$, + 1 D. cyl. axis 160° = $\frac{4}{10}$. One week later V. R. E. = $\frac{2}{10}$ − 3 D. cyl. axis 110° = $\frac{5}{10}$; L. E. V. = $\frac{3}{10}$ + 1 D. cyl. axis 10°

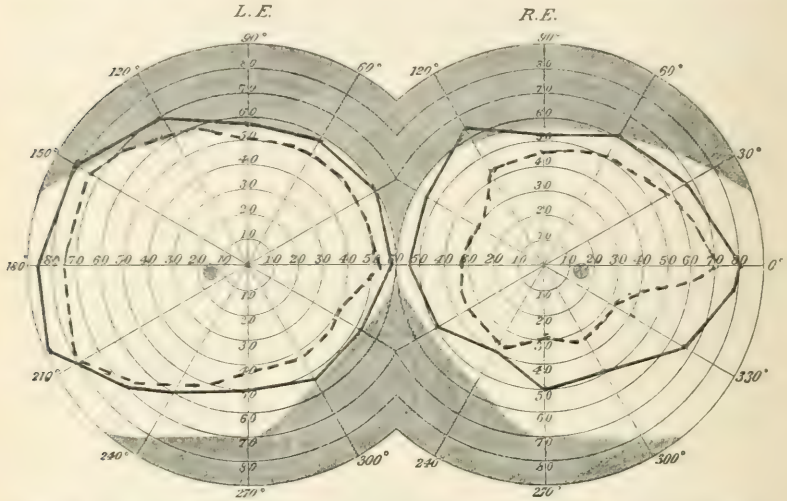


Fig. 2.—Dotted line, field before operation; solid line, after operation.

= $\frac{4}{10}$. With + 2.50 sph. added reads Snellen, 0.5. The refraction changed more or less, but I think has become fairly well fixed at the following (June 22d): R. E. − 2.50 D. cyl. axis 130° = $\frac{5}{10}$ +; L. E. − 1 D. sph. ⊂ cyl. − 1 D. axis 90° = $\frac{4}{10}$ +. This patient uses her eyes freely, and reads for hours at a time. Fields show considerable enlargement (Fig. 2). The appearance of the left eye is seen in Fig. 3.

CASE 4.—Miss M. H., aged seventy years. Referred by Dr. O'Connor, with the following history: Five years ago vision in the right eye began to fail; no pain at any time;

Fig. 3.—Appearance of eye after operation.

pupil dilated; lens opaque. No view of fundus obtainable. Tension + 2. Vision is reduced to perception of light. Left eye, central vision, $\frac{4}{60}$. Cupping of disc, slight opacity of lens, marked tortuosity of retinal vessels. Vision in the left eye improved under eserin to $\frac{6}{40}$, and the tension reduced to 42 mm.

On January 19th she came under my care, and I did an iridotasis, and she made a good recovery. An examination in June showed V. = $\frac{6}{40}$; tonometer, 28 mm. Decided increase in the visual field.

CASE 5.—Mr. D. F. C., aged eighty-six years. City hospital records, January 3, 1915. Twenty years ago noticed vision in both eyes began to fail. Soon after he was obliged to give up work. Patient has been a heavy drinker, but of recent years has been temperate.

In October, 1914, had a sharp attack of pain in his right eye that came like an explosion. This lasted several hours, and loss of sight followed for a time. Has had several attacks of a like nature since.

January 3, 1915. R. E. V. = fingers at two feet, conjunctival vessels injected, pupil dilated, anterior chamber shallow. Iris atrophic, cupping of disc. T. + 2, tonometer, 52 mm. Two days later operation of iridotasis. Fourth day after operation patient comfortable.

January 13th. No pain, eye looks clear, with the exception of a slight hemorrhage in the anterior chamber.

January 17th. Patient has been restless. Hemorrhage in the anterior chamber has increased. Kept in bed two days, and had hot fomentations.

January 21st. Hemorrhage has almost entirely disappeared.

February 9th. Discharged with complete relief and tension normal.

CASE 6.—Mr. F. F. N., aged eighty years. City hospital records, March 23, 1915. Eight years ago I saw him with chronic glaucoma, and advised an operation. Left eye, cataract immature. T. 40 mm. Operation of iridotasis. Patient recovered uninterruptedly, and vision improved slightly.

CASE 7.—Mrs. L. P., aged fifty-six years. December 26th, came into the hospital for pain in both eyes and loss of vision. Referred from the out-patient department with the following report: V. R. E. = fingers at three feet; T. +, beginning cataract. L. E., cataract; can see very little. Treated with eserin, but does not get much improvement. Both pupils somewhat contracted under the use of eserin. I operated on both eyes, with complete relief of pain and a gain of vision in the right to fingers at 20 feet with − 1 D.

In all the cases that I have operated on the relief has been prompt, and the patient has made an uninterrupted recovery, except in Case 5, but this was ultimately good, and in Case 2, where the iris was atrophic, but in this latter case the final result was good, as the patient has had complete relief from all pain after the successful operation for iridotasis downward.

DISCUSSION.

DR. F. H. VERHOEFF, Boston, Mass.: I have seen some of Dr. Harrower's cases, and have been very much impressed in favor of the operation. If the cases continue to do well, it seems to me that this is the operation that every one will be doing for chronic glaucoma, and that the ordinary trephine operation will be entirely given up. My first two attempts to perform the operation, however, turned out to be failures. One was in a case of chronic glaucoma in which a simple cataract extraction had been done many years before. I tried to carry out the operation as Dr. Harrower has described it, but found that the iris could not be pulled out intact. It was atrophic, and came out piecemeal. In the other case the iris prolapsed into the wound immediately, as one likes to have it do in performing an ordinary iridectomy. Perhaps this result was avoidable, but I think that when it occurs, it is impossible to carry out the procedure. I could not replace the iris properly, so I abandoned the attempt at iridotasis and did a sclerotomy by my own method and the result was satisfactory. The next case was perfectly successful. The case was one of intense glaucoma in a blind eye, and the eye was much congested. I thought this a

severe test for the operation, and the result was most satisfactory. I shall continue to use this procedure as a substitute for the trephine operation until I find some objection to it of which I am not at present aware.

DR. SATTLER: I should like to know whether Dr. Harrower has used the operation in cases of acute glaucoma?

DR. HARROWER: No, because iridectomy has been perfectly satisfactory in my hands in such cases.

DR. PETER A. CALLAN, New York: I am one of the older members of the Society, and my experience has been that, if you get an incarceration of the iris, you are apt to have dire consequences. Years ago, when we were doing simple extraction, the bad results did not appear until some time afterward. In other words, I do not think we can measure the result in these cases except by our findings years afterward. It may be only then that you get bad results. You have a constant source of irritation with this incarceration of the iris. Therefore I believe that it is premature to report these cases as successful until a sufficient time has elapsed to enable one to see whether any bad results will follow. It does not appear to me good surgery to incarcerate the iris, expecting not to have a bad result follow. I think that we ought to bide our time. It is all right to report the cases now, and the future will show whether the result is good or bad. I know, as one of the older members, that we were always sorry to have incarceration of the iris result from a surgical procedure. I do not care how you incarcerate it, you are going to have more or less traction and consequent irritation.

DR. CARL KOLLER, New York: I wish to say that I am in general agreement with Dr. Callan's view, and that, while I admire the enterprise of Dr. Harrower in practising this operation, I think that we have not had time yet to feel that it is successful. If you will allow me, I will make a few remarks on chronic glaucoma which I think are germane to the subject.

The relief of glaucoma consists in decompression—just as relief of tension in the skull consists in taking out a piece of bone, so relief of tension in the eye consists in taking out a

piece of the sclera. Both operations lead to success. I took up the use of the trephine very soon after it was first brought out, and have had a great many successes with it. I am very far from wishing to belittle the great service to operative technic and to humanity in general which Elliott has performed in devising and teaching his trephine operation. I was very enthusiastic about it in the beginning, but am not quite so enthusiastic as formerly. We are apt to publish our successes and not our failures, especially if they come late—a year or two later. With the trephine, you do not see how far you go, and you may go in too far and injure the lens. A posterior polar cataract, which seems to be the general reaction when injury is done to the lens, sometimes results. Therefore, if you injure the lens in any way at the operation you run the risk of getting posterior polar cataract, and, sometimes, later, complete cataract, even if the operation seems faultless and healing without reaction occurs. For this reason I have of late gone back to the former operation, that is, the Lagrange-Herbert operation modified by Holth, and described in the *Ophthalmoscope*, June, 1914. This gives excellent results, and is, in my experience and practice, superior to the trephine operation. After formation of a conjunctival flap as taught by Elliott, a special keratome is introduced 1½ mm. from the limbus into the anterior chamber, and then the central lip of the scleral wound is punched out with a special punch. The instrument that Holth has devised seems to me the best of the many that have been employed. If a posterior sclerotomy is done before the operation, as in acute glaucoma, it is not necessary to touch the iris, since there is no longer pressure to drive it forward into the wound. If, however, the iris presents itself, a complete iridectomy should be performed with Liebreich forceps. If it does not, after I have made the sclerotomy, I leave it alone. By this means one preserves a round pupil and has a soft eye.

Dr. D. B. Lovell, Worcester, Mass.: I should like to corroborate the work done by Dr. Harrower. I have seen him operate on most of the cases, and have seen a number of them some time after the operation was done. One case in particular I was very familiar with, and it has stood about

as long as any operated upon by him. It was the second case he did three years ago. The eye has been well ever since the operation, and the vision has been phenomenal. It was less than $\frac{20}{100}$ before the operation, and to-day, with a correcting glass, it is $\frac{20}{30}$, with a remarkably good field. The patient was absolutely blind in the other eye as the result of chronic glaucoma, and had been so for several years before the operation. In no case, so far, has there been the slightest irritation in any way, and the eyes have always been perfectly quiet.

DR. HIRAM WOODS, Baltimore, Md.: The point Dr. Callan has brought up merits careful consideration, for if an operation, devised for the relief of one danger, produces another, it is an argument against the operation. Is irritation as the result of incarceration of the iris as well founded as many think it is? I doubt it. I recall a case operated on fifteen or eighteen years ago. A boy had been struck with a stone and had a large iridodialysis. I did a scleral incision with a keratome, and drew the displaced iris into the wound, where it became fast. That boy was grown up and the eye was in perfectly good condition two or three years ago. I also recall a case of a large iridic hernia after simple extraction done in 1898 or 1899 in a man of seventy-odd years of age. He died only recently, and had normal vision all this time, with no irritation whatever. We see cases of anterior synechiæ that go on for years without irritation.

DR. D. F. O'CONNOR, Worcester, Mass. (by invitation): I was especially interested in one of the cases reported by Dr. Harrower. It was that of a woman of seventy, who had no unusual history except that five years previously she had noticed floating spots and shadows, with a gradual loss of vision. There was then absolute glaucoma in the right eye, and beginning glaucoma in the left; this was February 23, 1914. She continued under treatment for nearly a year; we used eserin solution chiefly. At the end of this time, however, she consented to an operation, and I referred her to Dr. Harrower. When first seen, vision was reduced to light perception in the right eye, and $\frac{4}{70}$ in the left. After operation the wound closed quickly, and all signs of irritation subsided rapidly. I followed the case since then, and

the tension, which at the time of operation was 40 mm. of mercury, has been reduced to 28, and has been at that point for three months. Vision has improved from $\frac{4}{60}$ to $\frac{6}{20}$.

The points that have impressed me in regard to the operation are, first, the absence of trauma; second, the increase of the visual field; and third, the lessening of the tension, as well as the improvement in vision.

DR. J. W. CAHILL, Worcester, Mass. (by invitation): The last case which Dr. Harrower has spoken of I happened to see first in the out-patient department of one of the hospitals. I distinctly remember the condition, because the patient had been getting atropin from an outside physician. She was absolutely blind, except for the perception of light, and had been so for ten weeks previously. She can now see large figures and letters. I saw four other cases he mentioned, at periods subsequent to the operations, varying from six months to one year, and in every instance there has been an absence of pain and irritation following the operative procedure. I wonder whether, in those injuries spoken of in which irritation followed, if it was not a corneal incarceration, whereas in the operative cases of Dr. Harrower it is entirely subconjunctival incarceration.

DR. DAVID HARROWER, Worcester, Mass.: In answer to Dr. Callan's question in regard to incarceration, I would say the iris is covered by the conjunctiva and other structures down to the sclera. As long as that remains covered you have good protection against any infection. I quite agree with Dr. Callan that we should not report these cases as successes. I should have said "temporary successes," because it takes so many years to find out whether our operations are successes from the point of view that Dr. Callan mentioned. They may be successful for two or three years, and then something comes up to destroy the result.

Regarding Dr. Koller's mention of my operation, I would say that it is not mine, but Dr. Borthen's, of Bergen, Norway.

Dr. Woods spoke of inclusion of the iris. I think that that is one of the important points. It is Borthen's theory that incarceration and traction, more than filtration, secure the results that he speaks of. Shortly after the Transactions of two years ago were printed I sent Dr. Borthen a copy of

my paper, and he wrote back an interesting communication, in which he said that he had done 206 operations since the last report, and that every one had been successful. This operation has been done, I suppose, for eight or ten years, and Dr. Borthen reports his results as successful.

TENOTOMY OF THE INFERIOR OBLIQUE MUSCLE.

WM. CAMPBELL POSEY, M.D.,
Philadelphia, Pa.

The credit of proposing division of the inferior oblique muscle to correct certain ocular deviations belongs to Landolt, for, so far as I have been able to ascertain, the first mention of such a procedure is contained in a paper published by this author in the *Archives d'Ophtalmologie* in 1885. After reviewing the anatomic relations and the physiologic action of the muscle, Landolt mentioned two conditions in which he thought division of its tendon indicated: (1) After paralysis of the superior oblique muscle, to counteract the loss in function of this muscle, the antagonist of the inferior oblique; (2) to prevent increase in myopia, by removing the pressure exerted by the inferior oblique muscle upon the venæ vorticosæ, thereby lessening the stasis in the veins of the uveal tract, which had been shown by Arlt to be a potent factor in the development of myopia. The communication was one of suggestion merely, and contains no mention of its trial by the author. Since its publication Landolt has apparently given no other public expression to his views, and the literature contains no further mention of the subject of tenotomy of the inferior oblique until Duane again brought it to the attention of the profession in a comprehensive paper before the British Medical Association in 1906.

As Duane, with characteristic modesty, permitted this communication to remain unpublished, I shall, with his permission, record from his manuscript, which he has kindly placed at my disposal, the indications which he gave for tenotomy of the muscle in question.

Indications for the Operation (Duane).

The operation may be indicated in any deviation due either to actual overaction (spasm) of the inferior oblique, whether primary or secondary, or to a condition in which, without there being any actual involvement of the inferior oblique, the diplopia and symptoms are such as would be produced by spasm of that muscle. The following are the conditions for which tenotomy of the inferior oblique may be required:

I. *Conditions Simulating Spasm of the Inferior Oblique.*

A. Paralysis of the superior rectus of one eye. Fixation with the other eye. No true spasm.

II. *Conditions of True Spasm or Overaction of the Inferior Oblique Secondary to Paralysis of Other Muscles.*

B. Paralysis of the superior rectus of one eye. Fixation with the same eye, causing secondary deviation of the other eye due to spasm of its inferior oblique.

C. Paralysis of the superior oblique or some other depressor or elevator with secondary spasm (contracture) of the inferior oblique in the same eye.

D. Paralysis of the abducens with compensatory spasm of the inferior oblique in the same eye.

III. *Primary Spasm of the Inferior Oblique.*

E. Spasm of the inferior oblique not due to paralysis of other muscles in the same or in the other eye.

Consideration of the Indicating Conditions. Varieties of Spasm and Pseudospasm of the Inferior Oblique.

A. *Paralysis of the Superior Rectus without Spasm of the Inferior Oblique of the Other Eye.*

These are cases in which the unparalyzed eye is used for fixation. Here we operate according to the principle of

Graefe that we can most satisfactorily compensate for the deficiency of movement in a paralyzed eye by operating on the associate in the sound eye, thus producing in the latter an artificial paralysis which weakens it in the same sense and for the same directions of the gaze that the natural paralysis weakens its fellow.

Why not advance the superior rectus in these cases? Often, of course, this would be the operation of election, but in some cases advancement is inapplicable because we are dealing with absence of nerve power. Here the operation would simply draw the eye up mechanically without helping it to move up in any normal manner. Again, in some congenital cases we may find, when we start to do an advancement, that the muscle itself is wanting or is replaced by a fibrous cord. Here, of course, advancement is out of the question and might even do harm.

Another objection against relying upon advancement in cases of marked superior rectus paralysis is that a very thoroughgoing advancement produces marked restriction of movement in the field of action of the antagonist; *i. e.*, in this case would produce in the lower field a diplopia which may be very annoying. Moreover, in such extensive advancements we can seldom regulate the effect with the proper accuracy, and the final result may differ in many degrees from the immediate one. Hence, in a case of very extensive deviation we shall get a better result if we first remove a large part of the deviation by a tenotomy of the contralateral inferior oblique, then correct the rest by an advancement, which, being only moderate in extent, will not hamper unduly the action of the opposing depressor and can be more readily regulated and controlled than if very extensive in amount.

B. *True Spasm of Inferior Oblique Secondary to Paralysis of the Superior Rectus in the Other Eye.*

This occurs when the eye with the paretic rectus is habitually used for fixation. In such a case the sound eye goes into a position of marked secondary deviation from overaction of the associate to the paralyzed muscle, the associate here being the inferior oblique.

This sort of secondary deviation occurs mainly when the paralysis is incomplete, often quite trivial. Fixation with

the affected eye is frequent in superior rectus paralysis, especially when the latter is traumatic or congenital, and may even occur when the eye used for fixation is the poorer sighted of the two. Slight congenital paralysis of this sort with secondary deviation of the other eye is quite a frequent condition. I have seen a number of such cases.

Referring to a case, the notes of which are appended, Duane goes on to say:

This case is a typical specimen of a group, the symptoms of which, constituting a fairly well-marked syndrome, may be stated as follows: In a case of right-eye paresis with secondary left-eye spasm we find, if the left eye is covered, a—(1) Moderate or often only a very slight restriction of the movement of the right eye up and to the right. (2) With both eyes open a marked upshoot of the left eye when the eyes are directed up and to the right. (3) Vertical diplopia with the image of the left eye below, this diplopia increasing fast as the eyes are carried up and to the right. (4) Tilting of the head toward one shoulder, usually the right. This is evidently done to obviate the diplopia. The attitude may be maintained so persistently as to cause an actual wry-neck. (5) The condition is not only congenital, but in some cases also appears to be hereditary.

If, as in the case cited, the disfigurement produced by the oblique movement is marked, and the diplopia is large in amount and causes some embarrassment of vision, and if there is a persistent wry-neck, tenotomy of the inferior oblique is indicated and would certainly be of service.

C. *Spasm of Inferior Oblique Secondary to a Paralysis of a Depressor or Elevator (Especially the Superior Oblique) in the Same Eye.*

In a case of this sort tenotomy of the inferior oblique would be resorted to only if (a) we were convinced that the condition is going to be permanent and stationary; (b) that the symptoms are such as to justify the operation.

D. *Primary Spasm of the Inferior Oblique.*

These cases are probably very rare. They should have one feature in common with the cases classed under category C, which distinguishes them from those classed under A. In

a spasm of the right inferior oblique due to a paresis of the left superior rectus, the left eye will fly up when turning to the left to make a parallel movement with the right eye, but it will not fly up when turning to the left to converge with its fellow, because in this action the left superior rectus is not specially called into play, at least as an elevator. In a spasm of the right inferior oblique, either primary or due to paralysis of a muscle in the right eye, the eye will fly up when turned to the left to perform either a convergent or a parallel movement. In such a case we may find binocular vision for distance combined with a marked vertical squint for near.

SUMMARY.

Tenotomy of the inferior oblique is indicated in—

A. Complete stationary paralysis of the superior rectus of the other eye, especially when this paralysis is traumatic or congenital.

B. Partial paralysis or even slight insufficiency of the superior rectus of the other eye when, owing to fixation being performed by the latter, the fellow develops a spasm of the inferior oblique so pronounced as to give rise to a disturbing (even if unnoticed) diplopia, a disfiguring upshoot of the affected eye, or a lasting torticollis. Such an indication is furnished especially by congenital cases.

C. In spasm of the inferior oblique, either secondary to paralysis of the superior oblique or some other muscle in the same eye, or else occurring as a primary affection, provided in either case the deviation and diplopia produced by the spasm are sufficiently disturbing to warrant an operation, and provided also that the condition gives good evidence of being stationary.

Having had occasion about the time Dr. Duane was engaged in the preparation of this communication to consult him about some details of a paper which I was myself preparing on the subject of congenital squint, and which was read before the Section on Ophthalmology of the American Medical Association in 1907, I first learned from him of the operation upon the inferior oblique and of the conditions in

which he deemed the procedure justifiable. I immediately began to perform the operation, and in the paper just cited gave the notes of five cases which had been subjected to it. Since that time I have performed tenotomy of the inferior oblique upon 12 other cases. The notes of these 17 cases, with those of four others kindly placed at my disposal by my colleague, Dr. Zentmayer, follow:

Case 1.—Female, aged twenty-six years, stenographer. For years past supraorbital headache, increased by near work. Left eye always the weaker. Add. good. R. H. 4°; esophoria 4°. Diplopia for far in connection with headache, associated with nausea. Pain more apt to be localized over O. S. Following correction prescribed and worn with comfort for three years: O. D. sph. − 1 D. ⌓ Pr. 1½° base down = $\frac{5}{5}$; O. S. sph. − 0.75 D. ⌓ cyl. − 0.25 D. axis 10° ⌓ Pr. 1½° base up = $\frac{5}{5}$. Examination showed marked overaction of R. I. O. when object carried to left. All other movements good except limitation of upward and outward motion in O. S.

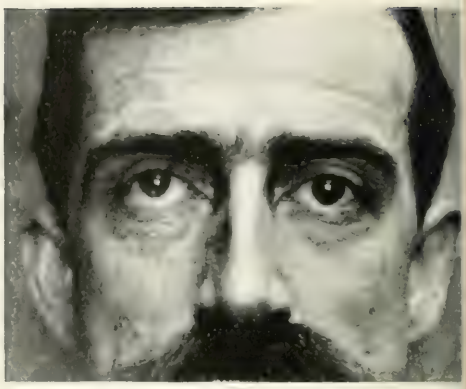
Diagnosis.—Congenital insufficiency of L. S. R. muscle with overaction of R. I. O.

Tenotomy of R. I. O. Immediately afterward, both eyes on same horizontal plane when looking to left. Now R. H. ½; exophoria ½. Orthophoria at near. Two months later orthophoria at all distances. No uptilting in O. D. No diplopia. Headache cured. Not seen since.

Case 2.—Female, aged thirty-four years. Formerly a stenographer. First saw double seven years before. Since then double vision has persisted with pain in O. D. Has worn for twelve years O. D. sph. + 0.37 D. ⌓ cyl. − 1.25 D. axis 10° ⌓ Pr. 4° base down = $\frac{5}{5}$; O. S. cyl. − 1 D. axis 170° ⌓ Pr. 1½° base up = $\frac{5}{5}$. With this correction, after close work, lines begin to slant, and diplopia occurs in the upper left field. Examination revealed some impairment of upward rotation in O. S. and up- and in-shoot of O. D. when objects directed to the left. R. H. 6°; esophoria 1°.

Diagnosis.—Probable congenital insufficiency L. S. R. with overaction R. I. O.

A



B

C

D

Fig. 1.—Case 3.—Insufficiency of L. S. R., with overaction of R. I. O. A. Primary positio eyes straight. B. Looking slightly to right; O. S. lags, O. D. fixing. C. Looking to left; O. I tilts up and in. D. Looking to left after operation; O. D. up and in, tilt much less pronounced.

Tenotomy R. I. O. Eyes now on same horizontal plane. Diplopia has disappeared. Pain in O. D. relieved, but R. H. 5°; exophoria 1½°. At end of four months, R. H. 5°; vertical orthophoria. O. D. sph. − 1.25 D. ⊂ cyl. + 1.50 D. axis 90° ⊂ Pr. 2° base down; O. S. sph. − 1.75 D. ⊂ cyl. + 2.25 D. axis 75° ⊂ Pr. 2° base up. Recurrence of pain. Advancement L. S. R. advised but refused.

CASE 3.—J. E. B., aged thirty-five years, casket maker, states that O. D. has been "crooked" when he looks to the left since birth. Frequently sees double for near, which interferes greatly with his work. Occasional dizziness. Claims vision good, both for near and far. No family history of strabismus. In primary position, eyes straight. Movements normal to the right. To the left, along the horizontal plane, O. S. fixes, O. D. tilts up and in. On upward motion, O. S. lags, particularly in motions up and out (Fig. 1). V. O. D. cyl. + 0.25 D. axis 145° = $\frac{5}{5}$; O. S. cyl. + 0.50 D. axis 115° = $\frac{5}{5}$. R. H. 18°; exophoria 2°.

Diagnosis.—Insufficiency of L. S. R., with overaction of R. I. O.

Tenotomy R. I. O., reducing up and in tilt of O. D. One week after operation, R. H. 8½°; exophoria 2°. Not seen since.

CASE 4.—Wills Hospital. Female, aged thirteen years. Left upper lid said to have drooped since birth. Left eye also turns out at times, and of recent years patient has exhibited a tendency to throw the head back to see clearly. V. O. D. = $\frac{5}{9}$; O. S. $\frac{5}{60}$. O. D. sph. + 1 D. ⊂ cyl. + 0.25 D. axis 90° = $\frac{5}{5}$; O. S. sph. − 1 D. ⊂ cyl. + 2.50 D. axis 90° = $\frac{5}{12}$. In primary position, O. D. fixes, O. S. turns somewhat down and out. Upward motion of O. S. very limited. Movements to the right normal, except O. S. on a lower plane; movements to the left, marked up- and in-shoot of O. D.

Diagnosis.—Insufficiency L. S. R., overaction R. I. O.

Under ether, L. S. R. advanced, R. I. O. tenotomized. Eyes now on same level in horizontal plane. Upward motion in O. S. still defective. Head straight.

CASE 5.—Female, aged seventeen years. (Previously reported, Case 9.) V. O. D. = $\frac{5}{6}$; H. + Ah. = 2 D.;

O. S. = $\frac{5}{5}$; H. + Ah. = 4 D. Fixing in median line, eyes are straight; object carried to right, O. D. lags and O. S. turns slightly up and in; object carried to left, O. S. rotates normally outward and O. D. deviates up and in; object carried up, O. S. fixes and O. D. diverges up and out, these motions being attended with marked wheel-motion in O. D.; O. D. goes slightly up under cover and diverges when patient looks up.

Diagnosis.— Insufficiency of L. S. R., with overaction of R. I. O.

Tenotomy of R. I. O. Now no deformity in primary position, but when eyes are directed to the limit of their rotary power to the left, O. D. still deviates somewhat up and in.

CASE 6.—Male, aged thirty years. (Previously reported, Case 10.) V. O. D. sph. + 0.50 D. = $\frac{6}{4}$; O. S. cyl. + 0.50 D. axis 180° = $\frac{6}{5}$; exophoria 12°; L. H. 20°.

Fixing in median line, O. D. fixes and O. S. deviates out and slightly up; looking to the left, both eyes fix normally; looking to the right, nystagmus appears; O. D. lags up and out, and O. S. tilts up and in; upward and outward movement in O. D. restricted.

Diagnosis.—Insufficiency of R. S. R.

Tenotomy of L. I. O. Reduced hyperphoria to 3°, lessening the upward and inward rotation of O. S., and giving marked relief to certain head symptoms and making the movements of the eyes freer.

CASE 7.—Male, aged seventeen years. Twitching of lids of left eye since seven years of age. Carefully refracted on several occasions by competent ophthalmologists, with but slight relief to symptoms; asthenopia. Examination showed marked nictitation of lids of both eyes, but most pronounced on left side. In primary position, eyes straight. Ocular movements good, but some short lateral nystagmic movements in each eye at end of lateral rotation. Also some up and in tilt to O. S. when eyes carried to right.

Diagnosis.—Insufficiency of R. S. R. muscle. V. O. D. cyl. + 0.25 D. axis 45° = $\frac{5}{5}$; O. S. cyl. + 0.37 D. axis 30° = $\frac{5}{5}$. L. H. 2°; esophoria 4°.

Tenotomy of L. I. O. One month later, no hyperphoria;

Case 9.—Insufficiency of both superior and right external rectus muscles.

Fig. 2.—Shows torticollis; head held in oblique position, right side of face presenting. Gaze is directed to the extreme left; O. S. is fixing and O. D. deviated up and in.

Fig. 3.—Shows almost perfect parallelism visual axes (O. D. slightly divergent) after ten omy of both inferior oblique and advancement right rectus externus.

Case 14.—Insufficiency of R. E. R. and L. S. R. Overaction of R. I. O.

Fig. 4.—Looking to right; eyes on same horizontal plane; external motion in O. D. restricted.

Fig. 5.—Looking somewhat to left and u O. D. goes up and in; external motion in O. normal; upward limited.

esophoria 6°. Eyes much more comfortable. Twitching almost imperceptible.

CASE 8.—Wills Hospital. Female, aged ten years. O. S. has squinted since infancy, with some drooping of left upper lid. No glasses. Diplopia complained of at all distances. O. D. fixing, O. S. turns down and slightly out. Upward motion limited in O. S. Other motions good. (Paralysis of L. S. R.) Up- and in-shoot of O. D. when object carried to the left, on horizontal plane, O. S. fixing. V. O. D. sph. + 0.50 D. ⊂ cyl. + 0.25 D. axis 90° = $\frac{6}{6}$; O. S. sph. + 1 D. ⊂ cyl. + 2 D. axis 90° = $\frac{6}{12}$.

Diagnosis.—Insufficiency of L. S. R. with overaction of R. I. O.

Tenotomy of R. I. O. Eyes brought to level. Diplopia abolished. O. S. still somewhat divergent. Advancement of L. S. R. advised but refused. Patient passed from observation.

CASE 9.—Male, aged ten years. (Previously reported, Case 6.) Good mentality; head well formed, but face underdeveloped; pronounced wry-neck; head held in oblique position, right side of face presenting. Dr. James K. Young reported contraction of left sternocleidomastoid muscle, with chin toward the right; left cervical, right dorsal, and slight left lumbar curve; slight hemiatrophy of left side of face. Primary position O. D., convergent strabismus, 45 degrees. Object carried to the right, O. D. fixing, O. S. deviates up and in; object carried to the left, O. S. fixes and O. D. deviates up and in; both eyes deviate upward under cover; marked limitation in outward rotation of O. D. (Figs. 2 and 3).

Diagnosis.—Insufficiency of both S. R. and of R. E. R. muscles.

Tenotomy of both I. O. muscles. Removed the upward and inward deviation of the eyes. Advancement of R. E. R. resulted in parallelism of visual axes; two months after operation no appreciable change in wry-neck.

CASE 10.—Wills Hospital. Female, aged five years. Eyes crossed since six months of age, O. D. mostly, O. S. at times. No family history of strabismus. On examination,

O. S. fixes, O. D. convergent 35° and turned slightly up. When O. D. made to fix, O. S. assumes similar converging position. Horizontal plane, movements to right, O. D. fixes and O. S. follows, but goes up and in at extremity of inward rotation. O. D. outward rotation good. Movements to left, O. S. fixes. Rotates externally normally. O. D. rotates up and in. Upward rotation of both eyes defective.

Diagnosis.—Congenital paresis of both S. R. muscles. Under atropin, V. O. D. sph. + 2 D. = $\frac{5}{9}$; O. S. sph. + 2 D. = $\frac{5}{7.5}$.

Division of both I. O. muscles under ether. Immediate relief to up and in tilt in lateral deviations in each eye. Residual alternating convergent squint of 20°. Correcting glasses prescribed. Six months later, eyes almost straight with glasses. Alternating convergent squint of 20° when glasses are removed. Not seen since.

CASE 11.—First consultation when aged eighteen months, with history of O. S. being convergent since birth. No family history of strabismus. Examination showed alternating convergent strabismus of 30°, O. D. being the preferred fixing eye. Upward motion in each eye limited. All other rotations good except marked up and in tilt imparted to opposite eye as object carried along horizontal plane to extreme right and left.

Diagnosis.—Insufficiency of both S. R. muscles, with overaction of both I. O. Five years later, condition the same. V. O. D. cyl. + 0.50 D. axis 90° = $\frac{5}{6}$; O. S. cyl. + 0.75 D. axis 150° = $\frac{5}{7.5}$.

Both I. O. divided under ether, correcting up- and in-shoot of each eye in lateral rotation. O. D. convergent 30°. Advancement of R. E. R. One month later, eyes practically straight. Six months later, alternating convergent squint of 15°. Corrected vision, now at age of seven years, normal in each eye.

CASE 12.—Male, aged ten years. (Previously reported, Case 28.) V. O. D. = $\frac{6}{3.5}$; H. + Ah. = 2 D.; O. S. = $\frac{6}{2.2}$; H. + Ah. = 3 D. O. D. deviates up and in, the deviation being more pronounced as fixing object is carried to the left; downward motion in O. D. limited, especially down and in;

no primary or secondary limitation or exaggeration of motion in left eye.

Diagnosis.—Insufficiency of R. S. O. with spasm of R. I. O.

Free tenotomy of R. I. O. relieved the tilting up and in, but eye still shows tendency to converge at times.

CASE 13.—R. F., stenographer, aged seventeen years. Mother first noticed that O. S. turned in when patient was aged two years. Glasses prescribed by competent oculist, without relief to squint, and vision in O. S. was noted as poor. Examination showed O. S. convergent 45° and slightly up. Upward motion increased when eyes turned to right. All motions in O. D. normal. External motion in O. S. defective, eyes being brought to within but 2 mm. of canthus. Downward motion in O. S. also limited, especially down and in.

Diagnosis.—Insufficiency of L. E. R. and L. S. O.

Advancement of L. E. R. No muscle found; capsule of Tenon advanced. Tenotomy of L. I. O. O. S. still convergent. Both I. R. carefully tenotomized, with resulting convergent squint in O. S. of 15°. Under atropin: V. O. D. sph. + 1.25 D. ⌒ cyl. + 0.50 D. axis 100° = $\frac{5}{5}$; O. S. sph. + 1.50 D. ⌒ cyl. + 0.50 D. axis 70° = $\frac{5}{8\,0}$. At end of one year eyes perfectly straight. No up and in tilt of O. S. Ocular movements free in all directions.

CASE 14.—Male, aged twenty years. O. D. squinted since birth. No family history of strabismus. No diplopia. O. D. tenotomized by an Italian ophthalmologist three years ago, but without cure of squint. Examination revealed marks of tenotomy over R. I. R. O. D. convergent 20° and turned somewhat up. In movements to right external motion in O. D. restricted, O. S. squinting in and somewhat up. In movements to left O. S. fixing, O. D. goes up and in. Normal external rotation in O. S., upward limited. When fixing with O. D. in median line, O. S. deviates up and in; when fixing with O. S., O. D. goes up and in (Figs. 4 and 5). V. O. D. = $\frac{5}{1\,5}$, with sph. − 0.50 D. ⌒ cyl. + 2.25 D. axis 70° = $\frac{5}{5}$; O. S. = $\frac{5}{9}$, with sph. − 0.50 D. ⌒ cyl. + 2.50 D. axis 100° = $\frac{5}{6}$ partly. Right hyperphoria, 8°; esotropia, 22°.

Diagnosis.—Insufficiency of R. E. R. and L. S. R. Overaction of R. I. O.

Tenotomy of R. I. O. with relief to up- and in-shoot of both eyes in movements in horizontal plane. R. H. = 2°; esotropia, 20°. O. D. still converges as before. Orthoptic exercise, followed by gradual straightening of visual axes. At end of three months, orthophoria at all distances, though O. D. still converges when correcting glasses are removed.

CASE 15.—Wills Hospital. Male, aged twenty-eight years. O. D. convergent since three years of age, following convulsions. Has been wearing high M. + Am. lenses since seven years of age. On examination, O. D. convergent 35° and turned slightly up. Outward rotation in O. D. limited, also in O. S. Upward motion in O. S. limited, especially up and out.

Diagnosis.—Insufficiency of both E. R. muscles and of L. S. R. muscle. Overaction of R. I. O. V. O. D. = $\frac{1}{60}$, not improved by high minus lens. Lens partially cataractous; O. S. = $\frac{2}{60}$ sph. − 9 D. ⊂ cyl. − 1 D. axis 170° = $\frac{5}{7.5}$.

Tenotomy of R. I. O. and advancement of R. E. R. One month later, O. D. still convergent 15°, but upward deviation has gone. Two months later, O. D. convergent 25°, but eyes on same horizontal plane.

CASE 16.—Wills Hospital. Boy, aged five years, said to have squinted since infancy. Several members of maternal side said to have had similar squint. Not verified. On examination, O. D. fixes, O. S. convergent 40°. Head abnormally large. Orbits appear shallow, oblique palpebral fissures. Horizontal plane, object carried to the left, O. D. fixes, O. S. turns down and out. Object carried to right, O. D. fixes, O. S. goes up and in. In upward motion, O. S. lags, especially out and up. In downward motion, O. S. deviates down and in. Monocular fixation O. S. Inward motion with O. S. good. Up and in poor, down and in good, outer good. Movements of eye not influenced by movements of jaw. Drs. Schwenk and Zentmayer concurred in diagnosis of paralysis of L. S. R. and overaction of L. I. O. in conjugate motions to the right. V. O. D. sph. + 1 D. = $\frac{6}{6}$; O. S. sph. + 1 D. = fingers at 1 meter.

Diagnosis.—Insufficiency of L. S. R. muscle with overaction of L. I. O. in conjugate motion to right.

L. I. O. divided. Upward and inward shoot of O. S. when object carried to the right is somewhat lessened. Eye drops down and slightly out as before in ordinary fixation. Object carried to the left, O. S. drops down and out. Six months later, in median line, eyes straight. Object carried to the left, O. D. fixing, O. S. goes down and out. Advancement of S. R. performed under ether, bringing eye upon same horizontal plane as O. D. O. S. still convergent 20°. Not seen after glasses prescribed.

CASE 17.—(Dr. Zentmayer's case.) S. W., aged twenty-seven years. Examination August, 1911. Glasses since fifteen; present pair six months. O. D., ptosis, lid covering one-fourth of cornea. In left lateral direction, O. D. turns strongly up in adduction. In left lateral direction, O. S. turns down in adduction. Orthophoria. R. H. 12°. Right esotropia.

Diagnosis.—Insufficiency of L. S. R., overaction of R. I. and I. O. muscles.

Atropin correction: O. D. sph. + 2.50 D. ⌓ cyl. + 0.50 D. axis 60° = $\frac{6}{10}$; O. S. sph. + 2.50 D. ⌓ 3° pr. b. down = $\frac{6}{5}$.

May, 1912, tenotomy of R. I. O. June 1st, R. H. 12°. June 14th, tenotomy of R. S. R.; next day, R. H. 5°. September 14th, with glasses containing in all 5° pr. bases up and down, R. H. = 5°. March, 1915, R. H. = 8°; Es. = 2°. Thinks eyes and health have steadily improved.

CASE 18.—(Dr. Zentmayer's case.) E. T., aged seventeen years. Examination April 4, 1903. States that O. D. has always tended to diverge on looking at near objects. O. D. cyl. + 0.62 D. axis 90° = $\frac{16}{12}$; O. S. sph. + 0.62 D. ⌓ cyl. + 0.50 D. axis 90° = $\frac{16}{13}$. Exophoria 1½° and 3°. Ab. 7°; Ad. 14°; L. H. 2½°. In primary position, simple exophoria. When eyes are turned to the left, O. S. continues to fix, but O. D. turns up about 10° when the eye becomes fully adducted. Right associated movements normal. On convergence above horizontal plane eyes diverge, O. D. first.

Diagnosis.—Insufficiency of L. S. R.; overaction of R. I. O.

September, 1910, tenotomy R. I. O., resulting in an

almost immediate relief of the irregular associated movements. October, 1911, still overaction of R. I. O. Exophoria = 7° for distance and 10° for near, but now has spontaneous deviation of O. D. but rarely. Formula given, combined with 2° pr. base in, O. S. September, 1913, seldom has headache and eyes are under control. O. D. cyl. + 0.75 D. axis 85° = $\frac{6}{5}$; O. S. sph. + 0.25 D. ⊂ cyl. + 0.75 D. axis 90° = $\frac{6}{5}$ (?)

October 14, 1914, orthophoria for distance; exophoria, 4° to 6° for near. No hyperphoria.

CASE 19.—(Dr. Zentmayer's case.) Wills Hospital. C. H., aged sixteen years. V. O. D. = $\frac{5}{3\,5}$; O. S. = fingers six inches. Eyes turned since birth. Partial coloboma of R. O. N. O. S., partial coloboma of O. N. and central choroiditis (coloboma?). Nystagmus. Conjugate deviation to right, nystagmus exaggerated. O. D. outward excursion somewhat limited; O. S. turns strongly up and in. To the left, O. D. follows normally; O. S. first makes a downward movement and then moves slowly on a horizontal plane toward the outer commissure; reaches median line at time O. D. is in extreme inward deviation. Outward excursion never full.

Diagnosis.—Congenital paralysis of L. S. R.

Tenotomy of L. I. O., bringing O. S. on same plane as fellow in conjugate deviation to right. Outward excursion increased. Advanced R. S. R; advanced L. E. R; advanced R. E. R. 7° of esoph. O. D. sph. − 2.50 D. ⊂ cyl. − 4.5 D. axis 90° = $\frac{6}{1\,0}$; O. S. sph. − 2 D. ⊂ cyl. − 4.5 D. axis 90° = $\frac{6}{1\,0}$.

CASE 20.—(Dr. Zentmayer's case.) Male, aged 19 years. O. D. sph. + 3 ⊂ cyl. + 1 D. axis 90° = $\frac{3}{3\,5}$; O. S. sph. + .50 ⊂ cyl. + 1 D. axis 90° = $\frac{4}{3\,5}$. Mentally insufficient. Born with O. D. turned up and in. Has double vision. Tower-shaped skull. In primary position, O. D. turned up and in. Looking to left, O. D. turns more strongly up. Lateral movements somewhat restricted.

Diagnosis.—Paralysis of L. S. R.

Tenotomy of R. I. O. Tenotomy L. I. O. and R. S. R. Still slight right hyperphoria. To the left O. D. still turns up by rotary motion.

Fig. 6.—Case 21.—Cerebrospinal syphilis with complete bilateral ophthalmoplegia interna and almost complete bilateral ophthalmoplegia externa. Secondary optic atrophy in both eyes. Right eye, complete paralysis of superior rectus, inferior oblique, internal rectus, and paresis of inferior rectus. Conservation of external rectus and superior oblique. Left eye, limitation of all movements of eye, especially below and to the nasal side. Marked overaction of inferior oblique when O. D. fixes. A. Fixing in median line slightly above horizontal plane; O. D. fixing, O. S. rotated up and in. Note faulty position of head; *i. e.*, tilted backward with face turned to the left, right side of face presenting. Corrugations on right side of forehead show overaction of frontalis in effort to overcome ptosis. Dilatation of palpebral fissure in ordinary fixation being due to stimulation of Müller's fibers. B. Looking to right; O. D. fixes, O. S. goes slightly up and in. C. Looking to the extreme left and slightly up; O. S. fixing, O. D. lagging behind. D. Looking slightly to the right; O. D. fixing, O. S. diverging. Shows pronounced ptosis in each eye, with overaction of right frontalis, head thrown slightly back.

CASE 21.—Male, aged forty years. Six years ago diplopia with drooping of lids following eruption on body. Vision also began to fail progressively. Antisyphilitic treatment inaugurated. At time of first examination by me ocular palsies were said to have remained unchanged during past four or five years (Fig. 6).

Fixes with O. D., O. S. being turned markedly up and in. When made to fix with O. S., O. D. turns down and slightly out. Pronounced double ptosis. In O. S. paresis of external, inferior, superior, and internal rectus muscles. Obliques alone seem conserved. In O. D. all muscles paretic, with exception of external and inferior rectus muscles. Vision reduced in O. D. to $\frac{3}{4\,5}$, in O. S. to $\frac{4}{5\,0}$, in consequence of simple atrophy of optic nerves.

Diagnosis.—Ophthalmoplegia externa with optic atrophy in consequence of cerebrospinal syphilis. Overaction of L. I. O. in consequence of paralysis of R. S. R. muscle.

Tenotomy of L. I. O. and L. S. R., reducing up and in deviation of O. S. at least one-half. One month later, eyes on same horizontal plane, and head, which had been held in oblique posture, now almost straight.

As indicated in the notes of the cases, all, with the exception of Case No. 21, were congenital.

The most common type is that illustrated by Cases 1, 2, 3, 4, 5, 6, 7, 8, 16, 18, and 19, and consisted of insufficiency of the superior rectus of one eye and overaction of the inferior oblique of its fellow. (Class II—B, Duane.)

The next most frequent type was insufficiency of both superior rectus muscles, with overaction of both inferior obliques, as illustrated by Cases 9, 10, 11.

Insufficiency of the superior oblique with spasm of inferior oblique of same eye (Class II—C, Duane) is illustrated by Case 12. The remaining cases are of mixed types, as follows:

Case 13.—Insufficiency of L. E. R. and L. S. O. Overaction of L. I. O.

Case 14.—Insufficiency of R. E. R. and L. S. R. Overaction of R. I. O.

Case 15.—Insufficiency of both E. R. and of L. S. R. muscles. Overaction of R. I. O.

Case 17.—Insufficiency of L. S. R. Overaction R. I. and R. I. O. muscles.

Case 20.—Insufficiency of L. S. R. Overaction R. and L. I. O.

Case 21.—Acquired external ophthalmoplegia, with overaction L. I. O., in consequence of paralysis of R. S. R. muscle.

The technic of the operation performed by Duane, and which I have followed, is the same as that suggested by Landolt, and consists in a division of the tendon of the muscle at its origin in the lower inner portion of the orbit. After injecting the tissues with a 2 per cent. solution of novocain, a curvilinear incision is made just superior and parallel to the lower and inner bony rim of the orbit, the skin and sublying tissues being divided with a scalpel down to the bone. A strabismus hook is now inserted into the wound and gentle traction made upon the tissues until the muscle is encountered. This is assured by the upward motion imparted to the globe by traction on the hook. The tendon of the muscle is then drawn forward into the wound and divided by a few snips of Stevens scissors. One or more stitches are inserted to close the wound, and a firm dressing applied. This latter is removed at the end of forty-eight hours, when the lips of the wound will be found to be firmly united, the only evidence of the traumatism of the operation being in some cases a subconjunctival ecchymosis of the lower, inner part of the globe, which disappears after a few days.

The results of the tenotomy were all more or less satisfactory. The up and in tilt imparted to the eye by the overaction of the muscle generally disappeared, and in a number of the cases immediate relief was experienced from the traction which the muscle had exerted upon the globe and from an annoying diplopia.

In the cases in which it was possible to ascertain the hyperphoria, prismatic measurement indicated that the tenotomy had reduced the deviation in amounts varying from 3 to 17 degrees. The lateral balance of the muscles was also improved, no doubt in consequence of the greater power given the adductors and abductors by the lessening of the vertical deviation. In none of the cases did any scarring of the lid follow the operation, nor did it produce any gross ocular deviation or interfere in any way with the proper action of the muscles that had not been disturbed.

I believe the operation a procedure of great value to meet the indications outlined by Duane, especially, however, in relieving the up and in tilt imparted to the eye by the overaction of the muscle in conjugate lateral movements where the superior rectus muscle of the fellow-eye is paretic.

In my paper on congenital squint I questioned the desirability of any form of operative procedure upon the eyes of children who had squinted from birth, until the age of at least ten years had been attained, as the permanent character of the ocular deviation is rarely fully established until that age. I now believe that it is wise to wait in some instances even longer, and to defer operation until five or six years later, as I have found in some of my cases, upon whom no operation was performed, that the development of the orbits, conjoined with the careful correction of the refraction error, which is often not inconsiderable in this class of cases, will at times render the muscular deviation negligible and the necessity for operative measures may be avoided.

As both eyes are employed in fixation and there is no danger of the development of amblyopia exanopsia, the necessity for early refraction is not as urgent in congenital as in concomitant squint. If there is considerable ametropia, however, I usually prescribe the correcting lenses, as determined by the retinoscope, at about three years of age. Orthoptic exercises are rarely of service.

DISCUSSION.

DR. ALEXANDER DUANE, New York: Of the indications for operation given in Dr. Posey's paper, I think the most important are, first, paralysis of the superior rectus, congenital or traumatic, with secondary deviation of the inferior oblique in the opposite eye; and, second, paralysis of the superior oblique, with secondary deviation of the inferior oblique in the same eye. The former condition is rather fre-

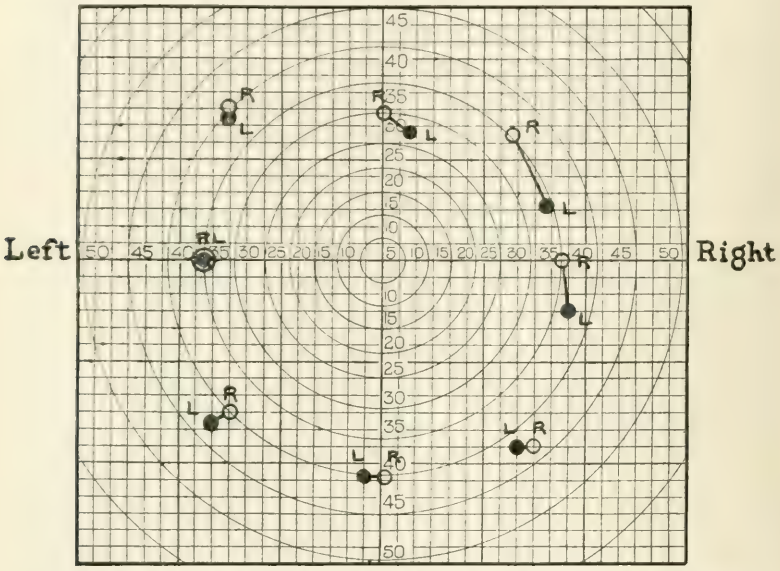


Fig. A.—Diplopia in congenital paralysis of the right superior rectus, with consecutive spasm of the left inferior oblique. Double images projected on a tangent plane at a distance of 30 inches. L, image of left (fixing) eye; R, image of right eye.

quent as a congenital anomaly. I think I have seen about 40 cases. The picture is characteristic, so that the diagnosis can be made in some cases even at the age of a few months. In a case of right-sided paralysis, for example, as soon as the patient looks to the right, the left eye shoots obliquely upward. The patient avoids looking to the right, and, to evade diplopia, either shuts one eye or turns the head to the

right and tilts it. In looking to the right, and especially upward and to the right, characteristic diplopia develops. This is well shown in Fig. A. The effect of operation on this diplopia is shown in the photographs (Fig. B). The characteristic attitude of the head, here carried to the point of actual wry-neck, is presented in Fig. B 1, while Fig. B 2 shows the effect of operation in relieving the deformity. The ocular origin of the torticollis in this case was demonstrated, first, by the fact that, when one eye was bandaged, the torticollis disappeared; and, second, that with the head in the in-

Fig. B 1.—Before operation.

Fig. B 2.—After operation.

clined position there was no hyperphoria, but when the head was held straight, a hyperphoria of about 16 degrees was apparent. This hyperphoria was almost completely relieved by operation. I may add that this patient had been treated by an orthopedic surgeon for a year without result.

Paresis of the superior oblique, with secondary deviation of the inferior oblique in the same eye, is another indication for tenotomy of the inferior oblique. The diplopia before and after operation in a case of this sort, in which the operation was supplemented by a tenotomy of the inferior rectus in the other eye, is shown in Figs. C and D. The result

here was quite satisfactory, and was exceedingly so in another case, in which a very disfiguring up-shoot, due to this cause, was relieved by tenotomy of the inferior oblique alone.

A word as to the technic of the operation may not be out of place. As indicated by Terrien, the incision is made at the

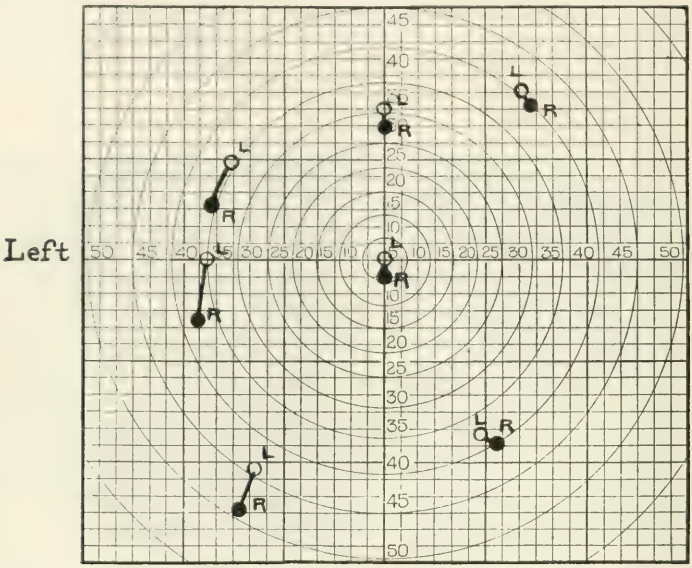


Fig. C.—Paresis of right superior oblique and spasm of right inferior oblique. Relation of double images. (Before operation.)

Double images plotted on a tangent plane at a distance of 30 inches. Circle (L) denotes image of left (fixing) eye; black dot (R), image of right eye. Note the vertical (right) diplopia, which is marked in the whole of the left half of the field of fixation and is about equally great in the upper and in the lower field (characteristic diplopia in left upper and left lower quadrants). Note also how, in both upper and lower fields, the diplopia decreases as the eyes are carried to the right. There is, however, no true continuously maintained single vision anywhere except perhaps in the extreme right portion of the field. In primary position, right hyperphoria of 4 degrees to 12 degrees, which, as the patient looks to the left, increases to 20 degrees. Right eye shows extorsion of 4.5 degrees.

intersection of the lower orbital margin with a perpendicular dropped from the supraorbital notch. It should be carried right down to and through the orbital septum, close to the orbital margin. In searching for the tendon, it is well to

keep close to the floor of the orbit and well to the inside, so as to grasp the tendon near its origin and also to avoid the aponeurosis of the inferior rectus, which may be connected with the inferior oblique by strong slips of fascia. The in-

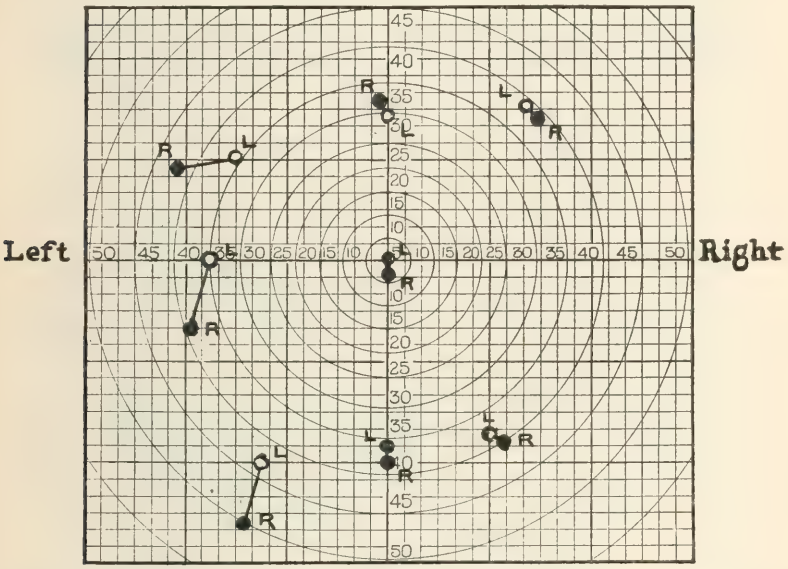


Fig. D.—Paresis of right superior oblique and spasm of right inferior oblique. Double images after tenotomy of inferior oblique.

Same patient as in Fig. C. Plot of double images taken five days after tenotomy of the right inferior oblique. Note that the vertical diplopia in the left upper quadrant is transformed into an almost purely lateral diplopia, while the vertical diplopia in other portions of the field is scarcely changed at all. This demonstrates the physiologic action of the inferior oblique, which acts mainly as an elevator, and whose elevating action is confined almost exclusively to the upper inner quadrant. Note further that the lateral diplopia produced by the operative paralysis of the inferior oblique is crossed instead of homonymous, as it should be, showing how little we can rely on the character of the lateral diplopia in diagnosticating paralysis of the elevators and depressors. Right hyperphoria in the primary positions till 5 degrees or 6 degrees, and still diplopia everywhere. Extorsion of right eye 0.

ferior oblique, more or less buried in fat and aponeurotic tissue, will be found if the hook is swept inward, with its point turned inward and upward.

The crude drawings, Figs. E and F, taken from dissections I have made, show the anatomy of the parts involved.

As to the age at which operation should be performed, I think that this depends entirely on the symptoms. If these are at all pressing, I should operate early—even at the age of three or four years. I should thus operate if—(*a*) the up-shoot is very disfiguring; (*b*) wry-neck has already developed; (*c*) a secondary convergent or divergent strabismus

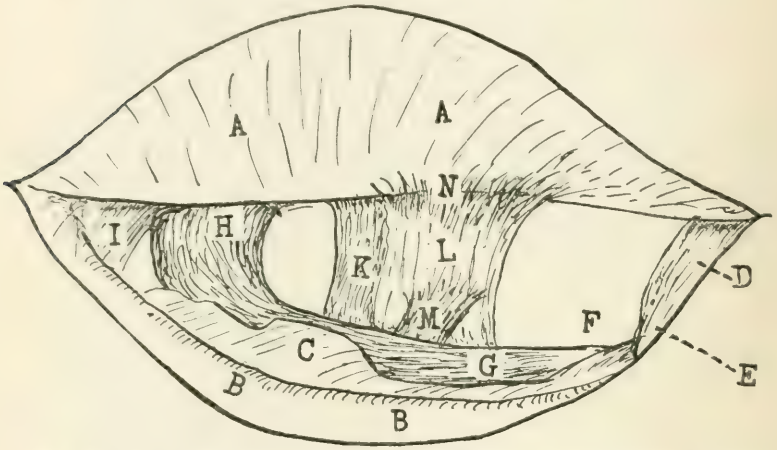


Fig. E.—A, A, cut lid-flap turned up and back, exposing orbital surface. B, B, bony margin of orbit. C, orbital septum, mainly cut away. D, lacrimal sac, and E, lacrimal duct, lying in front of C, and therefore not in the orbit (the remaining structures shown—G, L, etc.—lie behind C). F, tendon of inferior oblique, about 2 mm. long and situated at the point where Terrien's line (a line dropped perpendicularly from the supraorbital notch) intersects the lower orbital margin. G, belly of the inferior oblique, running almost horizontally outward in a mass of fat and fascia. At H turns abruptly backward, entering a fascial canal which lies directly beneath Tenon's capsule (see S, S, in Fig. F). I, deep expansion of fascia running to Tenon's capsule. K, inferior rectus muscle almost completely covered by a fascial investment, L, from which run a slip (M) to the inferior oblique and other slips (N) to the lower lid.

is developing before my eyes. This is a not infrequent concomitant of these paralyses, and is evidently due to the involuntary effort to get the disturbing double image out of the way. Of course, orthoptic exercises and correction of the refraction should first be tried in this as in other forms of strabismus; but, in my experience, it is just in these cases that these expedients fail and operation is necessary. In

my belief it is important in such cases to relieve first the vertical deviation, which is really the cause of the lateral squint; and in these cases, I would precede operation on the lateral muscles by a tenotomy of the inferior oblique.

DR. WILLIAM ZENTMAYER, Philadelphia: Merely because Dr. Posey has been kind enough to incorporate in his paper a history of my case do I rise to substantiate what he has

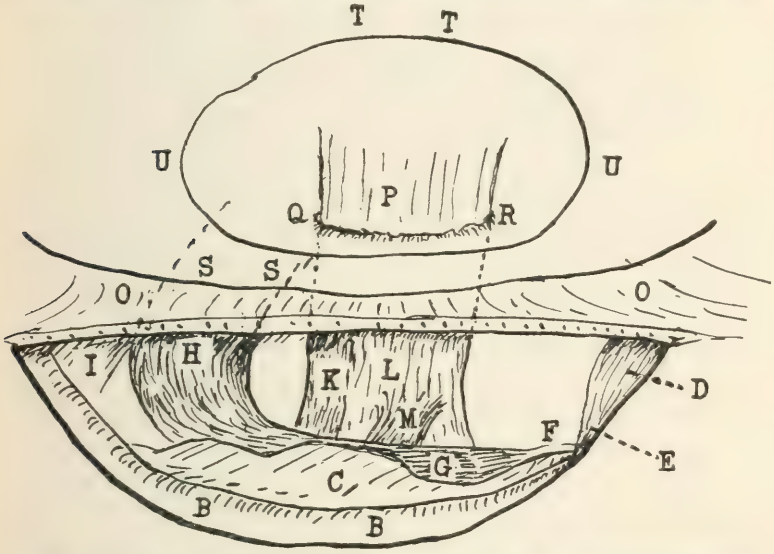


Fig. F.—B to M as in Fig. E. O, O, cut lid-flap held horizontally forward so as to show skin surface. P, intracapsular portion of inferior rectus tendon (very short and attached by very few lateral expansions to the globe). Pierces Tenon's capsule in a regular foramen, Q R, with puckered edges. In immediate relation with Q the inner margin of this foramen, and lying directly beneath Tenon's capsule is the inferior oblique muscle, here contained in a fascial canal (indicated by the dotted lines, S, S). T, T, U, U, cut edges of conjunctiva and Tenon's capsule (fused at T, T).

said regarding the ease with which the operation may be performed, the absence of scarring from it, and the beneficial results obtained. I shall not detail my cases. I simply want to say that in all the cases the immediate effect was very striking; but that in one the final result was not so good as the immediate result. Whether this was due to some fault of technic, so that reunion between the severed muscle

and the bone took place, or otherwise, I do not know. I think that the operation can be recommended particularly for cases in which there is paresis of the superior rectus muscle, and especially of the opposite eye. No one need hesitate to undertake the operation because of any danger in connection with it.

Dr. E. A. Shumway, Philadelphia: In a discussion of Dr. Duane's paper, read in 1911 before this Society, I put on record a case in which I had reduced the hyperphoria from 30 to 8 degrees by advancement of the superior rectus. Owing to the diplopia which the patient still had on looking upward, due to spasm of the inferior oblique of the other eye, I subsequently did a tenotomy of this muscle, with entire relief. A hyperphoria of 6 to 8 degrees still remained. At the time of operation there was some difficulty because of bleeding, and in doing the operation again I would add adrenalin to the cocain which is injected. The mixture that they use in Vienna, which consists of novocain or cocain in 1 per cent. solution nine parts, with one part of a 1:1000 solution of adrenalin, is an admirable one.

Dr. John E. Weeks, New York: I should like to know whether any particular effect on the action of the inferior rectus has been observed after these operations on the inferior oblique. The inferior rectus is so intimately connected with the inferior oblique by fibrous tissue that it would seem that traction by the inferior oblique would exert some influence on the action of the inferior rectus muscle.

Dr. Wm. Campbell Posey, Philadelphia: I have nothing further to add, except in answer to the question of Dr. Weeks. I did not notice any implication of the inferior rectus muscle in the 17 operations that I did. If there had been, I think that the preëxisting deformity would have been increased rather than corrected by tenotomy of the inferior oblique.

ROSACEA KERATITIS AND CERTAIN OTHER FORMS OF MARGINAL KERATITIS, NEUROPATHIC IN ORIGIN. TREATMENT BY PERICORNEAL NEUROTOMY.*

F. H. VERHOEFF, A.M., M.D.,
Boston, Mass.

As is well known, there sometimes occur associated with herpes facialis lesions in the central portion of the cornea. The most common etiologic factors in their occurrence seem to be acute coryza and gastro-intestinal disturbances due to improper food, but often no definite cause is apparent, owing, no doubt, to its trivial nature. These corneal lesions I have discussed somewhat at length elsewhere.† Not generally known is the fact that under the same conditions there may occur lesions of highly distinctive character limited to the periphery of the cornea.

Since it may now be accepted that facial herpes is neuropathic in origin and due to some irritative condition in the Gasserian ganglion,‡ it is altogether probable that these peripheral corneal lesions are likewise so. But there is another strong reason for believing them to be neuropathic; namely, their similarity to the central lesions of corneal herpes. In fact, they differ from the latter only in their situation and arrangement. They usually first appear as

* From the Massachusetts Charitable Eye and Ear Infirmary.

† *Jour. A. M. A.*, 1909, liii, p. 191; *Archives of Ophth.*, 1911, xl, p. 486.

‡ Pneumococci have been found in the Gasserian ganglion in cases of herpes, and recently Rosenow and Oftendal (*Jour. A. M. A.*, June 12, 1915, p. 1968) claim to have experimentally produced herpes of the skin in animals by intravenous injections of special strains of streptococci. The skin lesions did not contain the organisms, but the corresponding ganglia showed hemorrhagic lesions from which the organisms were recovered.

round, oval, or slightly irregular infiltrates immediately beneath the surface, and vary in size from a fraction of a millimeter up to several millimeters in diameter. According to the severity of the process, the epithelium may or may not remain intact over them, and they may or may not become slightly elevated, but they seldom undergo ulceration. They are stained by fluorescin in the same characteristic way as neuropathic lesions in general. As a rule, they are situated at a distance of about $1\frac{1}{2}$ mm. from the limbus, and, when multiple, often occur in a row running parallel with the limbus. Careful examination in the early stages shows that the larger spots are really made up of smaller ones, just as are the lines and spots in dendritic keratitis. This is most easily determined by staining them with fluorescin, and after washing away the stain from the conjunctival sac examining them quickly before the color has time to diffuse. The conjunctival congestion, which is most marked in the vicinity of the lesions, as well as the subjective symptoms of irritation, may be considerable or insignificant. Sometimes there are associated with the corneal lesions the herpetic nodules at the limbus previously described by me elsewhere. The duration of the process varies from a few days to a number of weeks. Vascularization of the cornea seldom occurs. In rare cases the lesions consist of vesicles from which filaments may sometimes grow.

Assuming the lesions to be neuropathic, their usual location at an approximately uniform distance of $1\frac{1}{2}$ mm. from the limbus plainly shows, it seems to me, that they occur at the terminations of the conjunctival nerves in the cornea. For it is known that the periphery of the cornea is supplied in part by nerve-filaments which extend into it from the conjunctiva. I have never been able to determine any reduction in the sensibility of the periphery of the cornea in these cases, but have attributed this to the fact that this portion of the cornea is normally rather insensitive and

that it is partly supplied also by the true corneal nerves. In exceptional cases, in addition to the lesions near the limbus, I have observed one or two spots as far as 3 or 4 mm. from the limbus. These I have explained by assuming that occasionally some of the conjunctival nerves extend unusually far into the cornea, and this assumption seems to be fully confirmed by the results of the operative treatment to be described.

The occurrence of these corneal lesions at the terminations of the conjunctival nerves is perhaps simply a manifestation of the general tendency of herpetic lesions to occur at the periphery of an area of nerve distribution. Thus, facial herpes is most apt to involve the lips or alæ of the nose. In any case it is necessary to assume that the particular ganglion-cells or nerves corresponding to these regions are especially vulnerable. Elsewhere I have suggested an explanation of the lesions of neuropathic keratitis in general, and this applies equally well to the marginal form under consideration. It is, briefly, that impulses from the affected ganglion-cells pass backward along the ordinary sensory nerves to the nerve terminals in the cornea, where they produce, by electrolytic dissociation, toxic substances injurious to the tissues. I have also described the histology of the lesions of superficial punctate keratitis which undoubtedly are essentially the same as those under consideration, and have shown them to consist of localized infiltrates of necrotic pus-cells situated immediately beneath Bowman's membrane.

In cases of rosacea of the skin, as is well known, there are sometimes marginal infiltrates or ulcers of the cornea.* In long-standing and severe cases these may repeatedly occur, and the cornea become superficially vascularized so that a more or less extensive pannus-like condition is finally

* In my experience rosacea keratitis is far more frequent than the rosacea conjunctivitis of Arlt, in which there are phlyctenule-like nodules at the limbus.

produced. The affected eye is usually extremely irritable, but the visual acuity is reduced only in the cases of pannus formation, usually the alcoholic cases. Fortunately, the lesions seldom become seriously infected with pyogenic organisms.

For several years I have regarded and treated rosacea keratitis as a form of neuropathic keratitis. My reason for this has been that careful examination of the lesions has convinced me that they are identical in nature with those of marginal neuropathic keratitis just described. Their situation is the same, their sizes, shapes, arrangement, and staining reaction with fluorescin are the same. The only differences are the longer duration and consequently greater tendency to vascularization of the rosacea lesions. These are differences of degree, not of kind, and may be attributed to differences in the agents producing the nerve lesions.

In long-standing cases of rosacea, especially the alcoholic cases with vascularization, the lesions may occur far out in the cornea. A possible explanation for this is that, owing to the long duration of and frequent recurrence of the process, nerve filaments from the conjunctival branches grow out abnormally far into the cornea.

If the foregoing conclusions are correct, and the corneal conditions described are really due to impulses passing along the conjunctival nerves, the lesions should promptly heal if the abnormal impulses are prevented from reaching the cornea. A simple method of cutting off the impulses would be to incise the conjunctiva in the region involved, that is, to do a partial peritomy. Within the past year I have done this in 15 consecutive cases with most successful and conclusive results. In every case healing promptly occurred. In 12 cases the corneal lesions failed to stain with fluorescin at the end of forty-eight hours. In the others they could be stained slightly for a day or two longer. These results seem to prove conclusively not only that rosacea keratitis

and the other forms of marginal keratitis described are neuropathic in origin, but that they are due to impulses reaching the cornea through the conjunctival nerves.

Four of the cases operated upon were cases of rosacea keratitis, in two of which there was vascularization. In one of the latter there had been recurring attacks for two years; in the other rosacea cases the durations were each about one month. The age of the patient in three rosacea cases was about fifty-three years, and in the other twenty years. The other cases were of the simple herpetic type. In three cases there was acute coryza and in two others herpes facialis. In the remaining cases the diagnosis was based chiefly upon the appearances of the corneal lesions alone, but was substantiated by the results of the operative treatment. In one of the herpetic cases the age of the patient was fifty-six years; in the others the ages were from seventeen to thirty-three years. The durations of the cases before operation as given by the patients were from three days to two months. In one case the lesions consisted of four epithelial filaments in a row attached to the cornea at a distance of 1¼ mm. from the limbus. Forty-eight hours after operation these had completely disappeared. In the other cases there were definite infiltrates, and these, of course, remained visible for some time after they could no longer be stained. The subjective symptoms of irritation promptly subsided in all but two cases. In one of these they persisted for twelve days in spite of the disappearance of the corneal lesions. In the other case the peripheral lesions healed promptly, but a dendritic figure appeared near the center of the cornea which healed in about a month under holocain treatment. This probably was due to a coincident involvement of the true corneal nerves.

The method of performing the operation that was used in most of the cases, and that I now employ, is as follows: The conjunctiva is incised at the limbus by means of scissors

for a considerable distance beyond the limits of the corneal lesions, care being taken to cut all the tissue down to the sclera. In some cases I have extended the incision over more than one-half the circumference of the cornea. The conjunctival flap is then undermined by snips of the scissors for a distance of 4 or 5 mm. back of the limbus, so as to sever as far as possible all nervous communications. The flap is then brought up to the corneal margin by means of two sutures, one at each end of the incision, as in the Van Lint operation for cataract. In the first few cases I also cut into the sclera at the limbus with a cataract knife, but later found this unnecessary. In one case I curved the conjunctival incision a considerable distance from the limbus and undermined the flap remaining. The result, so far as the corneal lesions were concerned, was perfectly satisfactory, but there was more conjunctival reaction than in the other cases. Perhaps this modification, however, should be given a further trial. Since the purpose of the operation is different from that of peritomy, and the technic is also somewhat different, it may be well to distinguish it by the term pericorneal neurotomy.

Whether or not the results of the operation will in all cases be permanent I am not yet prepared to say. None of my patients, however, has returned with a recurrence. It would seem probable that the severed nerves would regenerate, but their new distribution might, as a rule, be so widespread that no corneal lesions would result. It is obvious that the same condition might later occur in another part of the cornea or even in the same part, due to other cells of the ganglion becoming affected. The operation is so simple and free from danger, however, that there is no reason why it should not be repeated as often as indicated.

Rosacea of the skin is generally believed to be an angioneurotic condition due to some abnormal constituent of the blood. The reason for this belief is that the condition seems

to be primarily a dilatation of the vessels of the skin in certain regions. The acne lesions sometimes associated with it are now believed to be secondary and not essential to the process. Whether the affected vessels are dilated by stimulation or depression of the vasomotor nerves or the ganglion-cells does not seem to have been discussed. The question as to what the abnormal constituents of the blood are that cause rosacea apparently also has not been discussed. It is believed that improper diet is the most common cause, and we know that chronic alcoholism often gives rise to the condition. Judging from my own cases, tea drinking is a common cause. Possibly rosacea is not due to the presence of alcohol, tea, etc., in the blood, but is indirectly due to the chronic intestinal disturbances set up by them. As a result of the latter, incompletely digested proteins or other substances could be absorbed in abnormal quantities and produce deleterious effects on the nerves or ganglion-cells. That transient gastro-intestinal disturbances, although relatively slight, can affect the Gasserian ganglion is indicated by the experience of any one repeatedly subject to "cold sores."

Having shown, conclusively it seems to me, that the corneal lesions of rosacea are neuropathic, and assuming the skin lesions to be angioneurotic in origin, how may they be correlated? The corneal lesions may readily be explained by assuming that the same deleterious agent in the blood that produces the skin lesions, from time to time, acts injuriously on the Gasserian ganglion. In regard to the way in which this deleterious agent produces the skin lesions a number of possibilities suggest themselves. It might do so by acting upon the vasomotor endings, on the vasomotor centers, or upon the Gasserian ganglion. In the latter case the vasodilatation could be produced either reflexly by afferent impulses from the ganglion to the brain, or by efferent impulses from the ganglion to the skin. The latter

impulses could cause dilatation of the vessels either indirectly, by producing irritating substances at the terminals of the sensory nerves, or directly through the vasomotor nerves. This last possibility is supported by the recent experimental observations of Bruce,* which go to show that the nerves from the posterior root ganglia give off vasomotor branches at the periphery.

Obviously, the problem as to the explanation of the skin lesions of rosacea is a complicated one, difficult of exact solution. Since, however, the cornea has no vasomotor nerves, and the Gasserian ganglion must therefore be responsible for rosacea keratitis, the most reasonable assumption at present would seem to be that the skin lesions of rosacea are likewise due to irritation of certain cells in this ganglion.

DISCUSSION.

DR. T. B. HOLLOWAY, Philadelphia: In regard to Dr. Verhoeff's reference to acne rosacea, at the time of my own report, several years ago, I called attention not only to the ocular lesions, but to the various theories held in regard to the skin lesion. While I did not make a definite statement in reference to a possible neuropathic origin, I had this in mind, and, remembering the paper Dr. Verhoeff had presented on this subject a short time before, I looked over his article and found that he briefly referred to acne rosacea. At that time he did not definitely state his belief, but one could readily interpret his train of thought. If the operation he suggests will so promptly relieve the corneal condition or in any way relieve the tendency to recurrence it will prove of much value. I have not been fortunate enough to see as many cases of this character as Dr. Verhoeff.

I would like to call attention to other conditions that may give rise to changes similar to what he has described and which I believe to be of neuropathic origin. I believe that if we carefully examine the cases of polyarthritis that come under our observation we will find in a certain number these same peripheral punctate lesions occurring about 2

* A. N. Bruce: *Quarterly Journal of Exp. Phys.*, 1913, vi, p. 339.

mm. from the limbus. In using the term polyarthritis I am using it in the broadest sense, for, without other records, I cannot state at this time whether they occur in both rheumatoid arthritis and arthritis deformans of the Germans, or what Goldthwaite would refer to as osteoarthritis. Some two and a half years ago a patient with the latter condition affecting both eyes came under my observation with all the ocular manifestations of a typical neuropathic keratitis that was indistinguishable from the usual condition that may arise after removal of the Gasserian ganglion or avulsion of the sensory root. She was under observation in a hospital for several weeks under the usual treatment, including bandaging and the internal administration of various drugs, including thyroid extract, with little or no results. Dr. de Schweinitz may recall this patient, as he was good enough to see her with me. That was two and a half years ago, and at no time that she has been seen since then have I failed to demonstrate typical extensive lesions of each cornea. I then decided to examine a series of these cases, but for one reason or another have never completed the series. In all I examined 11 patients; several of these were under the care of Dr. Ralph Pemberton, who was carrying on some metabolic studies. Others were observed at the Orthopædic and the University hospitals. Of these 11 patients, 4 had definite corneal lesions, and, unusual as it may be, they form well-connecting links. One of these had only the peripheral punctate opacities that took the stain. Another had peripheral punctate opacities in one eye, while in the other there were punctate opacities with corneal changes characteristic of neuroparalytic keratitis that involved an irregular horizontal area of the cornea. The third was the patient first alluded to, while in the fourth patient there were peripheral punctate maculæ without staining. Contrary to Dr. Verhoeff's observation I found corneal sensation diminished in the three active cases. One of these cases was first recognized by the slow movement or lagging of the lid that occurs when winking. Time does not permit me to refer to some of the associated conditions frequently found in these cases of arthritis.

DR. F. H. VERHOEFF, Boston, Mass.: In the typical cases in which the lesions are confined to the periphery of the

cornea I have been unable to make out any hypesthesia. In one case, however, after the peripheral lesions had cleared up following the operation, a dendritic figure developed in the center of the cornea. Here the portion of the Gasserian ganglion supplying the true corneal nerves must also have been affected. In such cases hypesthesia of the cornea might well occur. Probably in Dr. Holloway's cases the ganglion was involved in this way. The lesion in the ganglion may not be of such a character as to produce central corneal lesions, but nevertheless cause hypesthesia.

SOME EXPERIENCES IN ENUCLEATION UNDER LOCAL ANESTHESIA.

WILLIAM M. SWEET, M.D.,
Philadelphia, Pa.

Every operator of large clinical experience has been compelled, in isolated instances, to employ some form of local anesthesia to remove an eyeball in a case where general anesthesia was considered to be too hazardous. The cocain was usually employed locally and by injection at the insertion of the recti muscles, and, after severing of the muscles, in the region of the posterior segment of the globe. These operations were far from painless, especially the severing of the ciliary nerves and the other tissues at the posterior portion of the globe.

In recent years enucleation under local anesthesia has been followed in most of the Continental eye clinics. The methods employed vary, but in all the usual local instillation of cocain in 3 or 4 per cent. solution is combined with injections back of the globe. The technic of these injections into the orbit differs, and may be summarized as follows: (1) Injection of a solution of cocain at the insertion of each of the four recti muscles, and, after the separation

of the conjunctiva at the limbus and the severing of the four straight muscles, injection in the region of the optic nerve of a quantity of Schleich's solution (Meyer). (2) Injection into the muscle insertions and in the region of the optic nerve by a curved cannula passed along the four straight muscles, or only along the interni and externi, of a 1 or 2 per cent. novocain and adrenalin solution (Siegrist). (3) Deep injection back of the eyeball, in the four meridians, of a large quantity of novocain and adrenalin solution, sufficient to cause exophthalmos and swelling of the lids (Seidel). (4) Injection of a 1 or 2 per cent. solution of cocain and adrenalin in the neighborhood of the ciliary ganglion, and also at the insertion of the four straight muscles (Elschnig).

In 1905 Meyer reported 80 cases of enucleation under local anesthesia, of which 46 had no pain, 24 complained of slight pain, and 10 had severe pain. Those who suffered pain were cases of chronic inflammatory glaucoma and severe iridocyclitis. He separated the conjunctiva and cut off the four recti muscles under simple instillations of cocain. After the structures were divided, he injected deeply into the orbit No. 2 Schleich's solution, and then divided the optic and ciliary nerves.

In 1906 Bruns and Robin reported 20 cases, the solution employed consisting of 10 drops of 4 per cent. solution of cocain, 10 drops of 1:1000 adrenalin solution, and 20 drops of normal salt solution. With a hypodermic syringe 10 drops of the mixture were injected along each rectus muscle deeply, behind the equator of the eye. The total quantity of cocain injected amounted to two-fifths of a grain. They found the method practicable, but not entirely devoid of pain. Robust and full-blooded men, above the average in weight, complained more of pain than the frail and delicate.

Siegrist recommended in 1907 a method of local anesthesia that he claimed to relieve all pain in enucleation. The

usual local anesthesia of the cornea and conjunctiva is secured by a 2 per cent. solution of cocain, and then a fold of the conjunctiva and capsule is grasped with forceps and the curved cannula of the syringe is passed close to the eyeball, as far as the entrance of the optic nerve and ciliary nerves, and $^3_4$ c.c. of a 2 per cent. solution of novocain with adrenalin is injected. This injection is made in the four meridians of the globe, $^3_4$ c.c. of the solution used each time. This method is extensively employed in the German eye clinics, using a solution of novocain and adrenalin, and making the injection to the nasal and temporal sides of the globe only. In Axenfeld's clinic the method is further modified by canthotomy before the operation is commenced.

Mende, of Siegrist's clinic, reported the result of 118 enucleations performed by the Siegrist method, of which 89 were painless, 23 complained of slight pain, and in 6 the pain was severe. Of these, 7 were under eighteen years of age, in 6 of which there was no pain, and 111 were over eighteen. Four patients vomited, but in no instance was there collapse. In a later report he includes 155 cases, 119 of which were painless, 28 slightly painful, and 8 had severe pain. Ernest Thomson, in a visit to the clinic at Berne, states that the operations he saw performed were absolutely painless. In the Siegrist clinic the orbital injection is made with a glass syringe holding 2 c.c., and this amount of a solution of 1 per cent. novocain, with a few drops of 1:1000 adrenalin solution, is injected by means of a slightly curved needle, two inches long, to the back of the globe, first from the nasal side, and a similar quantity from the temporal side. The syringe is entered along the lower border of the internal and external recti muscles, so as not to penetrate the muscles. A total of 1 c.c. of the same solution is then injected at the insertion of the four recti muscles. Thomson calls particular attention to the blanching of the skin of the upper and lower lids and the immediate neighborhood within

from five to ten minutes after the injection has been given, indicating that the anesthesia is complete. In the young and robust, a 2 per cent. solution of novocain is used. Seven and one-half grains of veronal are given the night before and one-half hour before operation.

Seidel, of Wagenmann's clinic, modifies the Siegrist method, and employs the same strength novocain and adrenalin solution, but uses a straight needle, 5 cm. long, and makes the injection into the orbit in the four meridians of the eyeball. The conjunctiva at a distance of about 4 mm. back of the eyeball is picked up, the point of the needle entered, and directed back into the orbit toward the middle point of a line between the optic foramen and the entrance of the optic nerve into the eyeball, and one-half of the contents of a syringe holding 2 c.c. of solution spent during the advancement of the needle. The other half of the solution is injected retrobulbarly. In a like manner the needle is inserted in the three other meridians, and the same quantity of the solution similarly injected. Immediately after completing the four injections there is considerable exophthalmos and edema of the lids. A compress is applied for twenty minutes, and then the operation is started. Anesthesia lasts for fifty minutes. The maximum dose of adrenalin is five to ten drops.

In 1908 Elschnig published his method of injection deep in the orbit in the region of the ciliary ganglion of a solution of cocain and adrenalin. The results, as summarized by Gradle, in 146 eyes removed by ciliary ganglion anesthesia, show 125 with no appreciable pain, 13 with moderate pain, and 8 with severe pain, requiring the use of a general anesthetic.

I first employed the Elschnig method early in 1912, and removed nine eyeballs by deep injections into the orbit, briefly reporting the results before the Section on Ophthalmology of the College of Physicians of Philadelphia. The

method of injection followed in these cases was that described by Löwenstein, of Elschnig's clinic. In most cases the injection was made with a Pravaz syringe, holding 1 c.c. of solution, 1 per cent. of cocain hydrochlorate in salt solution, with four drops of 1:1000 adrenalin solution to each syringeful.

The conjunctival sac is first rendered anesthetic by three instillations of 4 per cent. solution of cocain, three minutes apart. The external canthus is then stretched well outward, and the point of a rather thick needle, 5 cm. long, is entered close to the external orbital margin, along the lower border of the external rectus muscle, and pushed obliquely inward toward the median line until not more than ½ cm. of the needle remains visible. The point of the needle is now raised slightly, and one-half of the contents of the syringe injected. After waiting about half a minute pressure is made upon the ball, and, if the fluid has been injected in the proper place, the sensibility of a tender and inflamed eyeball disappears. The remainder of the contents of the syringe is then injected in the same situation. Should the eyeball still remain tender upon pressure after one-half of the solution has been injected, the needle is slightly withdrawn, pushed forward again in another direction, and the balance of the fluid injected. The needle is then withdrawn, the syringe again filled with the solution, and about four minims injected near the insertion of each of the straight muscles. In about five minutes the operation may be commenced. Some difficulty is experienced at first in passing the needle deep into the orbit, as the point strikes the external orbital wall, but upon withdrawing the point slightly and directing it more inward, it readily passes to the desired position. Before injecting the solution the syringe should be moved laterally to determine that the point of the needle is free and not caught in the optic nerve.

In the nine cases above mentioned there was practically

no pain during the operation in six, except in two instances, in which there was some tension on the orbital tissues in drawing the muscles forward with the strabismus hook. These cases were: Woman, aged thirty-four years, with pseudoglioma, eyeball removed for cosmetic reasons; man, aged fifty years, with shrunken and painful globe; man, aged eighty-two years, with inflamed, shrunken eyeball following injury some years previously; woman, aged sixty-eight years, with blind and tender eyeball following cataract extraction; woman, aged seventy years, blind, painful, glaucomatous eyeball; and man, aged seventy-eight years, shrunken and painful globe from old injury. One man, aged sixty-nine years, with slightly shrunken and tender eyeball from old iridocyclitis, complained of considerable pain when the optic and ciliary nerves were cut; and a man, aged fifty-six years, with extensive ulceration and staphyloma from severe burn, did not feel any pain when the tissues back of the eyeball were severed, but complained of severe pain when the collapsed eyeball was drawn forward to cut the optic nerve. The ninth case presented decided nervous phenomena following the orbital injection. In this patient there was also evidence of pain when the muscles and optic nerve were cut, but the patient's condition prevented injection beneath the tendons, as in the other cases, and the necessity for haste in removing the eyeball caused more than usual tension on the orbital tissues. The full history is as follows:

Woman, aged seventy-five years, patient of Dr. H. F. Hansell, Jefferson Hospital. R. E., simple glaucoma; L. E., blind from absolute glaucoma; deep injection of the ciliary vessels; T +1; no decided pain in eyeball, but constant, dull headache. Valvular heart lesion. She had a dread of general anesthesia, and asked that ether be not given. Operation performed in the general amphitheater of the hospital, and there was naturally not the quietness

that would have been secured in a small operating-room. After anesthesia of the conjunctiva with three instillations of a 4 per cent. solution of cocaine, the conjunctiva and capsule were grasped along the lower border of the external rectus muscle and the long needle of the syringe passed without difficulty back to the region of the ciliary ganglion. She complained of some pain during the entrance of the needle. After the needle had reached the desired situation one-half of contents of syringe was injected. After waiting about a minute, pressure was made upon eyeball, which the patient said was painful. Direction of the needle was slightly changed, and the remaining one-half of syringe injected. Immediately upon completing the injection of the last portion of contents of the syringe, and before needle could be removed, patient went into a convulsive seizure affecting the whole body, breathing stopped, and the face was deeply cyanosed. The finger was passed into the mouth, and the base of the tongue drawn forward and artificial respiration started, and inhalations of oxygen given. Breathing was soon established, the cyanosis disappeared, but she was still only partly conscious. No attempt was made to inject cocain beneath the muscle insertions, and Dr. Hansell immediately began the operation, and soon removed the eyeball. During the severing of the muscles, and when the optic nerve was cut, the patient indicated, by the spasm of the muscles of the face, that pain was felt. No primary or secondary hemorrhage. In about half an hour after being put to bed consciousness was completely restored. Healing was uneventful. No history of previous similar nervous attacks.

The nervous symptoms in this case were probably purely psychic, the result of the fear of the operation and the excitement natural in a large amphitheater in the presence of students. While it is possible to enter a vein and inject the solution directly into the circulation, the needle used was of rather large caliber, and had a large vein been entered, there would have been considerable hemorrhage into the orbital structures following removal of the needle, but none was found. The total amount of cocain injected was one-fourth

grain, an amount not likely to cause toxic symptoms. From the early experience of the general surgeons with cocain injections in minor operations, we know that toxic symptoms more frequently followed injections about the head than other parts, but any fraction of a grain of the drug was regarded as perfectly safe. When syncope did follow, it was regarded as due to fright, the sight of blood, or to the traumatism rather than to the toxicity of the cocain. The patient complained more than usual when the needle entered the orbit, and the nervous apprehension was probably more of a factor than the cocain in the rather alarming symptoms that occurred.

For a considerable period after the above experience I abandoned local anesthesia for enucleation, but later had a curved needle made, as recommended by Siegrist, but somewhat shorter, and employed the method of this surgeon of injecting a 1 per cent. solution of novocain with adrenalin at the insertion of the four straight muscles, and from 1 to 2 c.c. of the same solution in the region of the optic and ciliary nerves. In injecting the solution behind the globe the needle of the syringe is entered first along the lower border of the externus, and then along the lower border of the internus. This method was followed in 12 cases with satisfactory results. In nine the operation was practically painless—seven cases of iridocyclitis following injury, one case of panophthalmitis after cataract extraction, and one case of sloughing of the cornea. In two cases there was some pain, but in neither instance sufficient to cause the substitution of general anesthesia—one case of dense leukoma and one case of absolute glaucoma. Several of the patients complained of pain when the superior rectus was cut, due to the drag on the muscle by the hook. In one of the cases the conjunctiva was dissected up at the limbus before the injection was made back of the globe, as in the plan of Meyer, but a considerable portion of the solution escaped,

and the result was not as satisfactory as by the Siegrist method.

In none of the 21 cases reported was the primary hemorrhage greater than in enucleation under general anesthesia, and in fact the bleeding seemed to be less. In two there was slight secondary oozing sufficient to stain the dressings. In more than half of the cases a gold ball was implanted in Tenon's capsule after the removal of the eyeball.

In the few cases that had pain during the operation it was insignificant. I believe that many of the reported cases of severe pain are the result of undue traumatism on the part of the operator. There should be no pain in separating the conjunctiva, and little if any in cutting the muscles, providing there is no traction on the muscles with the hook. A considerable degree of the pain complained of when the optic and ciliary nerves are cut is undoubtedly the result of dragging on the orbital tissues. Not infrequently the scissors are thrust behind the globe before all the capsular attachments are divided, the back of the handles resting on the orbital rim as a fulcrum, and the eyeball almost pryed out of its position.

Mende, who has had a large experience in local anesthesia, gives as contraindications: Children and excitable people, in whom the psychic shock is more to be feared than that after narcosis; when the eyeball is badly damaged, so that the needle cannot be guided to the optic nerve region; in panophthalmitis, when the wound in the eyeball is opened; and in purulent conjunctivitis, on account of the risk of causing orbital abscess.

As Vail, in his résumé of the operation of enucleation under local anesthesia, well says, there should be perfect silence during the operation. The operator should avoid asking for sponges, scissors, or refer in any way to bleeding or shock, and if the patient shows apprehension, say a few

words of confidence to indicate that everything is progressing well.

It is hardly necessary to refer to the advantage that a satisfactory method of local anesthesia has over general narcosis in a definite number of cases. Where there is, however, no contraindication to general anesthesia, and the operator has a trained assistant to give the ether or chloroform, it is probable that in this country the older method will be the one most generally followed.

DISCUSSION.

DR. WALTER B. LANCASTER, Boston, Mass.: Perhaps my experience in enucleation under local anesthesia may be worth recording, for the reason that up to last winter I had done three or four such operations, and every one had been a failure so far as freedom from pain is concerned. In one case the patient behaved very well and I flattered myself that I had not hurt much, but I saw this patient a few months ago and asked her. She corrected my misapprehension very emphatically. The pain on cutting the nerve was of as severe and prostrating character as if she had been knocked down by a blow on the head. These operations were not done under ether because the patients had been told by some one never to take ether.

Last winter I became convinced that enucleation was being done in various places with results entirely different from these of mine, and that it must be a question of technic. Since January I have done, under local anesthesia, five enucleations, besides some tear-sac and other operations requiring dissecting, with most satisfactory results in every case, and at least three or four more done by the interns during that time have been equally successful. I have looked up the records of these cases. The first was a patient with an acutely painful staphylomatous eye following ulcer of cornea. He had a bronchitis which resisted treatment and made ether undesirable. Novocain, ½ per cent., and suprarenin, 1: 20,000, was used. Result: "Patient kept perfectly quiet and gave no evidence of feeling pain at any stage."

Another was an alcoholic—a poor subject for ether. His eye had been ruptured three days before by a heavy blow, and panophthalmitis was setting in—great hyperemia, chemosis, and proptosis. Patient objected to operation without ether, but consented when promised operation would stop and ether be given if he found it painful. Behaved well, and no trouble was experienced. After operation he said he felt some pain, but made light of it.

Another patient was a frail old woman in whom I thought evidence of toxic effect might appear. Pulse and general condition closely watched, but there were no bad signs whatever and no pain.

Summing up: Three of the five suffered no pain; two admitted some pain, but all onlookers were agreed that by their freedom from restlessness and from involuntary demonstrations of discomfort the patients showed that they were not suffering.

In all cases there was normal rapid convalescence. What were the changes in technic which accomplished so great a change?

First, I used some preoperative treatment, viz., morphin and scopolamin or atropin, sometimes bromid or a similar sedative, one to two hours before operation.

Second, I convinced myself that I had been unnecessarily cautious, say rather timid, about possible toxic effects. There is no doubt that cocain is an uncertain drug, far more toxic to some than to others. Though I have never had or even seen a case of dangerous poisoning, I have had a keen dread of the possibility, for, of course, I have read of them. When I found that surgeons were using 1 or even 2 per cent. novocain in quantities as large as 50 or even 100 c.c. in various parts of the body, I felt it was unwarranted to be afraid to use one-tenth to one-twentieth as much, especially as the nature of the operation was such that a considerable quantity of the injected solution would escape during the operation and by no means all be absorbed. I therefore determined to use novocain more freely. In the first case I used ½ per cent. and injected about 5 c.c. I decided that a stronger solution in smaller quantities would be more effective, with less trauma, so later used 1 to 1½ per cent., combined with suprarenin 1:15,000, and *I injected enough to produce the result desired.*

If during operation the patient noticed some discomfort, 1 or 2 c.c. more were injected where needed.

Third, sufficient time is allowed after injection for the effect to be produced. To inject and immediately begin work is allowable in the Schleich (infiltration) method, but not when the method of producing anesthesia is by the effect of the drug on the nerve through absorption, rather than through pressure.

Fourth, I avoid injecting more than a few drops in one place without moving the needle, so as to be sure that the injection is not by any chance intravenous. In this perhaps I am again unnecessarily cautious.

Fifth, it is well to operate as gently as possible, avoiding vigorous tearing or other unnecessary force, either with closed scissors or with hook. There is no need for haste, and gentle manipulations are, of course, less painful and require less anesthetic to control their perception.

Small as my experience has been, I feel satisfied that with a good quality of drug and proper technic one can count on producing completely satisfactory local anesthesia. As to its safety, that is another matter, but since no bad symptoms were detected in any of my cases, and since surgeons are daily using ten to twenty times as much for a single operation under conditions where absorption can take place, and yet few bad results are reported, I feel that my former caution was excessive and unwarranted.

Lastly, as to effects on the healing, my cases are far too few to permit me to speak with any degree of finality. No bad effects were seen in any of my cases, although some of the patients were not very good surgical risks.

I believe that I shall use local anesthesia freely in the future. I should say very freely, except that I think in gas-oxygen we have a method which will become more popular as it is better known, and so reduce the field where novocain is the method of choice.

DR. W. K. BUTLER, Washington, D. C.: It may be interesting, in connection with Dr. Sweet's paper, to recall the limited experience that I have had, consisting of two cases in which enucleation was done under strictly local anesthesia. In one case there was a painful, shrunken globe, with detachment of retina in the other eye. The patient experi-

enced considerable pain, due, no doubt, to faulty technic. The other case was one of election, as the man did not want to have general anesthesia. He also suffered. My experience has led me to think that it is a good thing to supplement this method by giving a few whiffs of gas and oxygen, as Dr. Lancaster suggests, after the preliminary dissection. In the one case in which this was used, where the patient had a severe heart lesion, contraindicating general anesthesia, it was very successful, the combination of the two methods rendering the operation painless. This is the only experience that I have had in the few cases that did not lend themselves well to general anesthesia.

MASSIVE GRANULOMA OF THE SCLERA (BRAWNY SCLERITIS), WITH THE REPORT OF AN UNUSUAL CASE.*

GEORGE S. DERBY, M.D.,
Boston, Mass.

PATHOLOGIC EXAMINATION BY F. H. VERHOEFF, M.D.,
Boston, Mass.

In 1897, under the title of "Brawny Infiltration of the Conjunctiva and Sclera," Schlodtmann[1] added to the knowledge of scleritis in describing what is undoubtedly in clinical appearance and pathologic features a special disease, differing markedly from the anterior nodular scleritis so commonly seen in young adults affected with tuberculosis. Cases had previously been reported by Baumgarten[2] in 1876 and by Schirmer[3] in 1895, but neither had recognized the distinctive character of the disease. Since Schlodtmann's three cases, one of which was examined under the microscope, the following cases, all with pathologic examinations, have been reported: Friedland,[4] Uhthoff,[5] Parsons and

* From the Massachusetts Charitable Eye and Ear Infirmary.

Spicer,[6] Prochnow,[7] Wagenmann,[8] Roosa and Oatman,[9] Bietti,[10] Komoto,[11] Verhoeff,[12] Gilbert,[13] and Fleischer[14]; and besides these a number of others are on record where the clinical picture suggested the disease, but where no satisfactory pathologic examination was obtained. Various other cases, notably those of Gayet, Coppez, Donald Gunn, Brailey and Kostenitsch,[15] have been placed in the same category, but careful consideration of them shows that in all probability they do not belong here.

Brawny scleritis, renamed annular scleritis by Parsons,[16] and also known as gelatinous scleritis, usually appears typically, and in the classic case, as a gelatinous, brownish-red infiltration of the conjunctival, episcleral, and scleral tissues, most marked close to the limbus. The succulent swelling often overhangs the cornea and extends back a variable distance from the limbus, even to the equator and farther. In the diseased area there is enormous dilatation of the blood-vessels and lymphatics. Pain and tenderness are acute. As the process advances the cornea is involved in the form of a deep, interstitial opacity, always advancing until no clear tissue remains. This infiltration is accompanied by the formation of deep and also superficial blood-vessels. As the disease extends anteriorly, so also it penetrates into the deeper membranes of the eye, and almost invariably the uvea takes a greater or less part in the process. Synechiæ, corneal precipitates, vitreous opacities, and even retinal separation may be found. The sight fails, pain becomes acute, and finally the globe comes to enucleation. The progress of the disease is characterized by its extreme chronicity and by its acute exacerbations and remissions.

Although the majority of cases reported are of the classic type first described by Schlodtmann, it is now recognized that the clinical picture may vary according to the severity of the process and to the part of the eyeball principally affected. Thus, the changes may be most marked in the

posterior half, as in the case of Wagenmann and in the one here reported. The uveal involvement may be severe or nearly absent, the retina may show extreme changes with marked separation, as in Fleischer's case, or be comparatively little affected. Pain is usually a marked and distressing feature, coming on in severe attacks. Tension is most often increased, but may be diminished, according to the stage in which the disease is seen. The red, succulent swelling is characteristic, but by no means always present, and in many cases the diagnosis is made only after enucleation has been performed. Again, in some instances, the early picture cannot be distinguished from that of anterior nodular scleritis.

The prognosis is grave, as the eye usually comes to enucleation on account of pain and loss of sight. In a large majority of cases but one eye has been affected; in three, however, the sight of both eyes was practically lost (Schlodtmann, Uhthoff and Prochnow), while in two more (those of Verhoeff and Baumgarten) the second eye was mildly affected.

Etiology.—The etiology of this disease is still an open question. In the majority of cases no definite cause could be made out. There are but few definite facts to be found if we analyze the cases.

Gilbert believed gout to be the cause, as he found lesions of that disease elsewhere in his patient. Against his view it may be pointed out that in no other case have gouty manifestations been evident, that the pathologic examination of his case does not support his view, and that in the case to be reported here examination of the blood for uric acid showed a normal content.

Bietti believed his case to be due to tuberculosis, and points to some suggestive features in the histologic examination to bear out his contention. In the discussion of Bietti's case, Axenfeld was able, on the basis of a case of his own, not pathologically examined, to give some support to this

view. Against tuberculosis, however, lies the evidence of all the other reported cases and that of the microscope. Tubercle bacilli, though sought for, have never been found, and in no case has a positive focal reaction to tuberculin been obtained.

The microscopic picture gives more support to a diagnosis of syphilis than to any other disease, and yet in only one case, that of Verhoeff, was the Wassermann reaction positive. In our case and in those of Gilbert and Fleischer it was negative. The only evidence in favor of syphilis is the microscope, and that is inconclusive. A positive Wassermann reaction can be regarded as of little more importance in the diagnosis of a local lesion of this nature than is a general reaction to tuberculin without focal manifestation in the eye. The cause of brawny scleritis is still undetermined.

Age.—Basing our statistics on the sixteen pathologically examined cases, we find the age by decades as follows:

AGE	CASES
Thirty to forty	2
Forty to fifty	2
Fifty to sixty	2
Sixty to seventy	4
Seventy to eighty	6

Thus, the average age of the sixteen cases is sixty-one and a half years. The oldest case is that of Verhoeff, seventy-six years; the youngest is ours, thirty-four years.

The following case is presented: F. S., female, teacher, single, age thirty-four years, of Fitchburg, Mass.

F. H., mother, had Bright's disease. Maternal aunt had pulmonary tuberculosis.

P. H., sickly child, measles, mumps, and scarlatina. Since sixteen years of age has been in good health. Best weight three years ago—134 pounds.

P. L., five years ago she had an attack of inflammation in the right eye (said to have been scleritis), which lasted

one month. Two and one-half years ago the eye again became inflamed, and it has never become entirely normal since. During this time she has had considerable pain in eye and forehead. Early in July, 1914, she came to Boston to obtain advice as to her eye, and was seen by Dr. Myles Standish, who suspected a tubercular condition and referred her to Dr. J. B. Hawes, 2d, for general examination.

Dr. Hawes reported: weight, 114 pounds; hemoglobin, 85 per cent.; heart and lungs negative, except for faint systolic murmur. No glandular enlargement. Pirquet mildly positive. General condition poor, but nothing definitely abnormal found. Urine negative.

On July 14th she came to the infirmary and was admitted on the service of Dr. Verhoeff, with the diagnosis of scleritis. Examination of the right eye showed considerable injection of the ocular conjunctiva, some of the vessels being quite large; cornea clear; pupil regular, large, inactive (under mydriatic); anterior chamber normal. Tension normal. Ophthalmoscope shows vitreous opacities large and small. View of fundus cannot be obtained. Vision, $\frac{2}{200}$, not improved. The left eye was normal. Vision, -1.50 sph. $= \frac{20}{20}$.

Urine, 1020, no albumin or sugar. The Wassermann test was negative at this time and in December. She did not react to 1 mgm. or to 10 mgm. of old tuberculin. After a second injection of T. O., 10 mgm., the temperature rose to 99.8° F., and she felt a little out of sorts. There was no noticeable reaction, however, in the eye. Ten days after entrance in the hospital she developed a couple of small nodules in the conjunctiva. She remained in the hospital until September 14th without any definite change in her condition.

In August a second course of diagnostic injections of T. O. gave a second negative result.

On October 27th she was referred to the Tuberculosis Class and came under my charge. At this time there was no essential change in her condition, there was no nodular thickening of the sclera, and the eye did not suggest a tuberculous scleritis. What was especially noticeable, however, was the very marked, I may say enormous, enlargement and tortuosity of the episcleral veins. The cornea showed a small, localized opacity; the tension was sub-

normal (8 mm.); there was a descemetitis, and the fundus could not be seen on account of vitreous opacities. During treatment in the class for six weeks the case remained a puzzle. She was put on general building-up treatment, and reported once a week.

On December 3d a separation of the retina was detected downward, and transillumination over this area was faulty, although light was transmitted well over other portions of the sclera bordering on the limbus. A diagnosis of probable intraocular new-growth was made, and the patient was advised to have the eye removed. She acquiesced, and enucleation with implantation of a glass ball in Tenon's capsule was performed the next day. Healing took place without incident.

When the eyeball was sectioned and the nature of the disease became evident, in view of the recent findings of Gilbert and of Verhoeff in cases of this nature, it seemed desirable to use every effort to ascertain whether signs of either syphilis or gout were present.

On December 29th the patient was readmitted to the hospital, and on the following day blood was taken for a further Wassermann and for uric acid. Following this, she was put on a meat-free diet for twenty-four hours, and then was given 100 grams of sweetbread. Twenty-four hours later blood was again taken to be tested for uric acid. Our thanks are due to Dr. J. H. Pratt, under whose supervision the uric-acid tests were made. Both tests for this substance were within normal limits, the second specimen showing 2.2 mgm. The Wassermann was also negative. January 8th the patient was discharged and soon returned to her work.

PATHOLOGIC EXAMINATION BY DR. F. H. VERHOEFF.

The globe, including the thickened sclera, measures anteroposteriorly 30 mm., horizontally 26 mm., and vertically 26 mm. Globe sectioned vertically.

The abnormal size and shape of the globe is found to be due to an enormous thickening of the sclera. This is greatest in the lower outer quadrant posterior to the equator, where it forms an ill-defined nodule, 20 mm. to 27 mm. in diameter and 13 mm. thick. The thickening is thus greatest

in the vicinity of the inferior oblique muscle, but no trace of this muscle is found in the specimen. The sclera, however, is everywhere else greatly thickened except for a few millimeters immediately behind the limbus. The thickened tissue is very firm and slightly grayer in appearance than normal sclera, so that anteriorly the comparatively normal sclera can be seen to split up into bundles and lose itself in the pathologic tissue.

The cornea shows a faint old corneal opacity near the limbus and several punctate deposits on its posterior surface below; otherwise it is normal. After fixation, the anterior chamber is found normal in depth and free from coagulum. The filtration angle is not blocked by the iris. The pupil, 4 mm. wide, is round and free from membrane. There are two pigment spots on the lens capsule (artefact?), but no definite synechiæ. The iris is not noticeably thickened. The lens is *in situ* and clear. The vitreous chamber is reduced in size and is misshapen, owing to its compression by the thickened sclera (Fig. 1). The retina is separated almost everywhere, but remains adherent in places, especially above and in the vicinity of the disc. The irregular subretinal spaces are filled with clear serum. The vitreous is semifluid.

The ciliary body is everywhere greatly thickened, in some places to 2 mm. There is no typical cyclitic membrane, but the surface of the pars plana below is coated with a firm, white layer about 1 mm. thick. The choroid is also greatly thickened. The thickening is greatest anteriorly (1.5 mm.) and gradually becomes less until near the disc it disappears. Within the choroid are numerous, round, gray spots. The optic disc is swollen. The nerve-stem is intact, but on the outer side the dural sheath is involved in the overgrowth of tissue in the same way as the sclera.

Histologic Examination.—Fixation in 10 per cent. formalin, followed by a saturated solution of potassium dichromate, twenty-four hours; also by Levaditi's method for spirochætes, and Marchi's fluid. Celloidin embedding. Staining by a variety of methods.

The pathologic process is found to involve chiefly the sclera, the changes within the globe evidently being secondary. The sclera is everywhere affected, but least so near

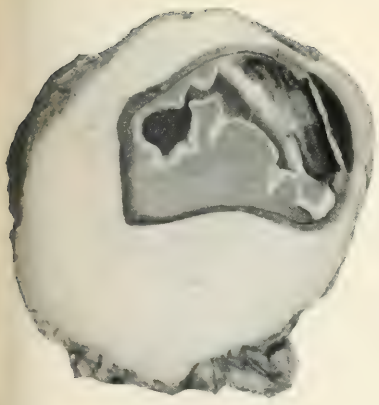


Fig. 1.—Massive granuloma of sclera, showing enormous thickening of sclera. Globe sectioned in vertical plane passing through outer third.

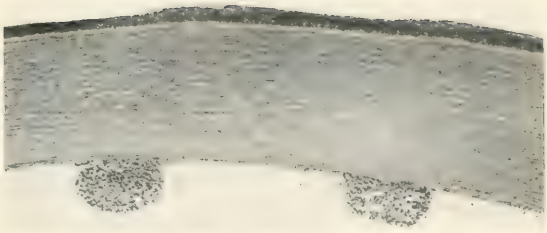


Fig. 2.—Massive granuloma of sclera. Precipitates on posterior surface of cornea. (Photo × 35.)

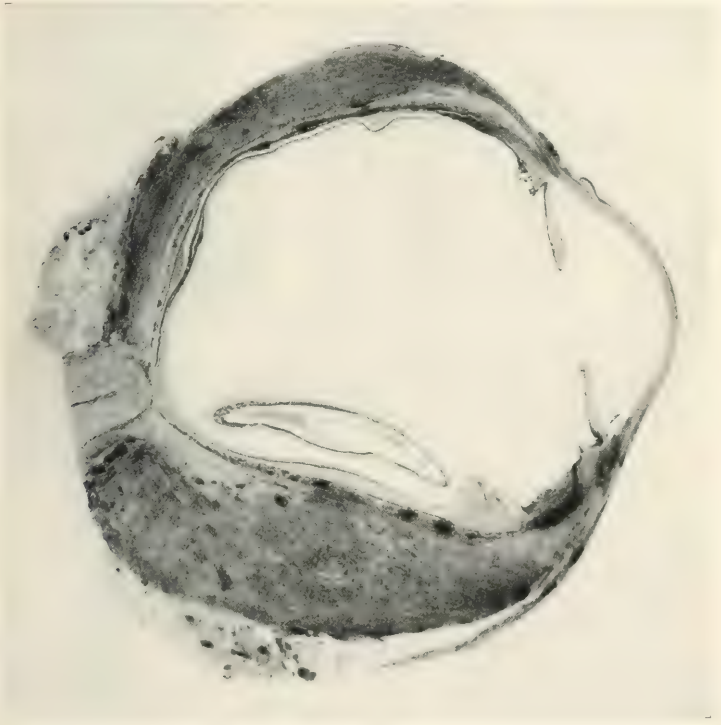


Fig. 3.—Massive granuloma of sclera. The lens has been removed. Note lymph-follicles in choroid and orbital tissue. Also exudate on pars plana ciliaris below. (Photo × 4. Reduced one-fourth.)

the limbus, where it is comparatively normal. As the sclera is followed backward its bundles are found to be more and more split apart by granulation tissue densely infiltrated with cells. These cells are almost exclusively plasma cells, and where the interstitial masses are small, the granulation tissue is difficult to recognize. The granulation tissue is giving rise to dense fibrous tissue, so that the extraordinary thickness of the sclera finally reached is due partly to an exuberance of fibrous connective tissue and partly to masses of granulation tissue and plasma cells between the connective-tissue fibers. In the thickest portion the fibrous tissue predominates. Within the cell masses occasional eosinophiles and a few lymphocytes are also seen. No giant-cells are present. Colloid globules, left by degenerated plasma cells, are abundant, and are brought out with especial prominence by the Gram stain. The new tissue contains no elastic elements that stain differentially.

The new tissue is not encapsulated at the periphery, but is rather sharply limited. Nevertheless, in places where orbital tissue has been left attached, the same inflammatory process can be seen extending into it, and the orbital tissue also shows isolated lymph-follicles. Near the disc, only the outer layers of the sclera are much involved. On the outer side of the nerve the dural sheath has been invaded by the inflammatory process extending from the sclera.

Within the new tissue occur here and there large lymph-follicles containing definite germinal centers and a few endothelial phagocytes. These are most numerous near the conjunctival surface. A marked feature of the specimen is the endarteritis of the blood-vessels. It is difficult to find a single artery in the body of the tissue that is not completely obliterated or collapsed. The thickening of the intima is due to proliferation of spindle cells, the nuclei of which stain feebly. It is evident, therefore, that the endarteritis is of a slow type. In the depth of the tissue even functionating capillaries are infrequent, and dilated veins are absent. A few small areas are found considerably infiltrated with pus-cells, the largest being 0.25 mm. in diameter. In several sections an area of this kind shows definite caseation, the stroma staining deeply in eosin and the pus-cells showing chromatolysis and fragmentation of their nuclei. The

Marchi specimens do not happen to contain any of these areas, and show only a few fat-droplets within the granulation tissue.

The conjunctival and episcleral tissue, a small amount of which was left at operation, shows moderate edema and marked infiltration with plasma cells. The veins are here greatly dilated. Some of the arteries show slight endarteritis, but in general the conjunctival vessels are free from intimal proliferation.

The cornea is normal except near the limbus, where the inflammatory process has extended for a short distance into its deeper layers. Below, stretches of granulation tissue and plasma cells extend between the lamellæ for a distance of $1\frac{1}{4}$ mm. from the canal of Schlemm. On the posterior surface of the cornea there are a number of large precipitates apparently identical with those described by Fuchs in serous uveitis (Fig. 2). They are composed almost exclusively of large mononuclear cells, with round, or sometimes indented, nuclei, and many of them contain pigment. These large precipitates contain almost no plasma cells, but toward the limbus below there are increasing numbers of plasma cells adherent to the endothelium. The ligamentum pectinatum is densely infiltrated with plasma cells everywhere, and below the filtration angle is filled with these cells.

The root of the iris is beginning to adhere to the ligament below, and is densely infiltrated with plasma cells. Elsewhere the iris stroma shows relatively little infiltration, and except for slight edema is otherwise normal. Adherent to and possibly arising from the anterior surface of the iris are numbers of mononuclear cells, some of them pigmented, which here and there form small clumps. There are no synechiæ.

The ciliary muscle is everywhere highly edematous and infiltrated to a greater or less extent with plasma cells, but the ciliary processes are almost normal. There is no ordinary cyclitic membrane anywhere, but on the surface of the pars plana there is a thick layer of new tissue which ends abruptly at the ora serrata. It consists of a dense mass of fibrous tissue, between the fibers of which are large collections of plasma cells, and contains a few blood-vessels, mostly of capillary size. A few small arteries showing

obliterative endarteritis are seen within it. Beneath it the epithelium in places shows proliferation and in others complete disintegration. The tissue closely resembles the new tissue in the sclera. Probably it has resulted as a reaction against the plasma cells which had collected on the surface of the ciliary body here. Above there is a marked exudation of plasma cells through the epithelium of the pars plana, but no formation of membrane.

The choroid anterior to the equator is highly edematous, the tissue spaces and subchoroidal space being distended with serum. It is also markedly congested, densely infiltrated with plasma cells, and here and there shows large perivascular nodules of lymphoid cells. At the level of the ora serrata, both above and below, it is invaded by granulation tissue extending into it from the sclera. Posteriorly it is less and less involved, and near the disc is practically normal.

The retina shows no marked changes. It is moderately edematous, and anteriorly shows perivascular infiltration with lymphoid and plasma cells. The ganglion-cells are still present in considerable numbers. The optic disc is highly edematous. The nerve-stem is apparently normal.

Bacteriologic Examination.—The Levaditi method shows no spirochætes or other organisms. Sections stained for tubercle bacilli in carbolthionin and by the Gram method give negative results. No sporothrices can be found in sections stained by Mallory's connective-tissue stain.*

REMARKS.

The pathologic process in this case may be briefly described as a slow invasion of the sclera by granulation tissue extremely rich in plasma cells, associated with an abundant new formation of dense fibrous tissue. Within the new tissue are a few small areas of purulent infiltration and caseation. A marked feature is obliterative endarteritis. The involvement of the intraocular structures, while well marked, is relatively slight compared to the scleral process, and is

* B. S. Wolbach (personal communication) has recently found that this method stains the organisms well in sections of experimental sporothricosis.

clearly secondary to the latter. The partial separation of the retina is no doubt due to transudation of serum from the edematous and infiltrated choroid.

The process is thus similar to, if not identical with, that found by the writer in brawny scleritis, differing only in degree. In the present case the new tissue is much more abundant, whereas the necrosis is insignificant when compared to that in Schlodtmann's and Friedland's cases of brawny scleritis. It is possible, however, that the necrosis is always due to the obliterative endarteritis. In the very early cases of Parsons and Oatman necrosis was not observed at all.

Judging by the histology of the lesions the inflammatory process in this case is also identical with that described as endothelioma of the optic nerve sheath by Parsons.[17] Examination of sections of Parsons' case* shows the same exuberant growth of fibrous tissue, the same plasma-cell infiltration, the same extreme endarteritis, and the same kind of involvement of the sclera and dural sheath of the optic nerve as in the present case. It shows also numerous large lymph-follicles, which differ from those in the present case only in the fact that their germinal centers are more sharply defined. Possibly it is the undifferentiated lymphocytes within these that Parsons regarded as endothelial cells. His diagnosis of endothelioma, however, was only a tentative one, and the writer can find nothing in the sections in his possession to substantiate it. Clinically, Parsons' case differs somewhat in the situation of the inflammatory process, but there is no apparent reason why the same process should not chiefly involve or begin in the posterior part of the sclera or even in the dural sheath of the optic nerve.

As to the cause of the condition, the pathologic examina-

* By chance, three sections of Parsons' case given out as class specimens at the Royal London Ophthalmic Hospital were available for study and direct comparison with those of the present case.

tion of the present case furnishes no conclusive evidence. It would seem, however, to exclude tuberculosis and all the known infectious processes except syphilis. In this particular case the massive formation of fibrous tissue somewhat suggests a fibroma, but the marked chronic inflammatory reaction associated with it would seem to rule out this possibility. Still another possibility is that the process represents a scirrhous lymphoblastoma. In fact, from the histologic examination alone this cannot be definitely excluded, although it is, *a priori*, improbable that such a tumor should arise in the sclera.

For a more detailed discussion of the pathologic lesions of this disease, reference is made to Verhoeff's article. From his examination of sections from four previously reported cases, those of Schlodtmann, Friedland, Parsons, and Roosa-Oatman, he concludes that the scleritis is the primary lesion, while the involvement of the other ocular tissues takes place secondarily, and the present case furnishes no ground for changing this view.

This case is characterized by a chronic inflammatory process in the eye lasting over a period of five years. The clinical picture was that of a rather mild scleral inflammation with an accompanying uveitis, and in the later stages an extreme engorgement of the episcleral vessels, even more marked than that ordinarily seen in absolute glaucoma. The appearance of the eye suggested a high tension, but it was always soft to the finger, and the tonometer readings were a low normal or below the normal limit. In spite of the low tension there was no sign of shrinking. On the whole, pain was not marked. The pathologic examination showed a relatively enormous scleral thickening composed of new fibrous tissue, granulation tissue, numerous plasma cells, with a few small areas of necrosis and caseation and marked obliterative endarteritis. The changes were most

marked in the posterior half of the eyeball. Pathologically, the changes found in every respect resemble those found in brawny scleritis, although the scleral thickening is much greater than in any case previously described.

The only other case in which the posterior half of the eyeball was more markedly affected than the anterior is that of Wagenmann, described as posterior scleritis. Wagenmann, however, recognized that the lesions described by him were characteristic of brawny scleritis.

Perhaps the enormous production of fibrous and granulation tissue in this case, in contradistinction to the others, was due, in part at least, to the patient's age, thirty-four years, whereas the average is sixty-one and a half years.

An especially interesting feature of the case lies in the fact that, although in the classic case of brawny scleritis, as first described, the disease affects by choice the anterior part of the eyeball and reaches backward to the equator and forward into the cornea, here the part of the sclera least involved was that adjacent to the limbus, except in the region below the cornea.

A study of the cases of brawny scleritis leads to the conclusion that the clinical picture of the disease must be enlarged to cover a much wider range than that originally described by Schlodtmann. It will vary according to the seat of the disease anteriorly or posteriorly, according to the severity of the process, and according to the degree in which the uvea takes part. Verhoeff makes the interesting observation that the massive formation of fibrous tissue in this case somewhat suggests a fibroma, but that this diagnosis may be ruled out by the marked inflammatory reaction.

In this connection, an observation in the literature by Saemisch[18] is of interest.

Female, aged ten years. For two years loss of sight; for six weeks blindness and pain in eye and head. Only in this last period was a change in the appearance of the eye noted.

O. S., slight swelling of lids, several large and tortuous veins in conjunctiva of globe, deep subconjunctival injection at limbus, cornea slightly cloudy, anterior chamber shallow, iris discolored and greenish, pupil enlarged and presents a gray-yellow reflex. A mass presenting two prominences seen behind the lens, one prominence on the right and one on left, but they meet in vertical meridian. Tension + ; 18 mm. of protrusion; movements curtailed. Vision nil. Diagnosis, intraocular tumor, enucleation. No recurrence in two and one-half years.

Pathologic Examination.—Retina totally separated. In region of papilla, retina and choroid grown together and form a 2 mm. thick mass. Subretinal space filled with a yellow, cheesy mass, made up of round cells. From the entrance of nerve to the inner side extends a thick tumor, 10 x 8 x 5 mm., intimately connected with the sclera, which looks normal. In the region where the retina and choroid were grown together there were several areas of round cells. The tumor consisted of fibrous tissue and cellular elements between the fibers. Diagnosis, fibroma of sclera and fibroid degeneration of the retina and choroid.

It is not impossible that this case is one of brawny scleritis. Certainly, the inflammatory reaction here would seem to rule out a simple fibrous tumor.

My excuse for presenting this communication, based on a single case, lies in the great rarity of the disease, its obscure causation, and on the fact that it has not previously been discussed before this Society.

REFERENCES.

1. Schlodtmann: Ueber sulzige Infiltration der Conjunctiva und Sclera, *Archiv. f. Ophthal.*, 1897, vol. xliii, p. 56.
2. Baumgarten: Ein Fall von sclerosierender Keratitis, *Archiv. f. Ophthal.*, 1876, vol. xxii, Abt. 2, p. 185.
3. Schirmer: Zur pathologischen Anatomie der Skleritis und Episkleritis, *Archiv. f. Ophthal.*, 1895, vol. xli, Abt. 4, p. 158.
4. Friedland: Zur pathologischen Anatomie der Skleritis, *Archiv. f. Ophthal.*, 1899, vol. xlviii, p. 283.
5. Uhthoff: Weiterer Beitrag zur pathologischen Anatomie der Skleritis, *Archiv. f. Ophthal.*, 1900, vol. xlix, p. 539.
6. Parsons and Spicer: *Trans. Ophth. Soc. of U. K.*, 1902, vol. xxii, p. 306.

7. Prochnow: Zur Klinik und pathologischen Anatomie der sulzigen Scleritis, Inaug. Diss., Greifswald, 1902.
8. Wagenmann: Zur Kenntnis der Scleritis posterior, *Ber. d. Ophthal. Gesellsch.*, 1903, p. 140.
9. Roosa and Oatman: A Case of Unilateral Annular Scleritis, *The Post-Graduate*, 1905, vol. xx, p. 216.
10. Bietti: Sull Anatomia patologica della Sclerite e delle sue complicazione, *Annali di Ottalm.*, 1908, p. 178; *Review Annals of Ophthal.*, 1909, p. 379; and *Jahresbericht*, 1908, p. 185.
11. Komoto: Beitrag zur pathologischen Anatomie der Skleritis, *Klin. Monatsbl. f. Augenh.*, 1909, vol. ii, p. 761.
12. Verhoeff: Brawny Scleritis, *The Ophthalmoscope*, January, 1913.
13. Gilbert: Zur Aetiologie und pathologischen Anatomie des sulzigen Skleritis, *Archiv. f. Augenheilk.*, 1914, vol. lxxvi, p. 111.
14. Fleischer: Ueber anatomischen Befund bei einen Fall von schwere Skleritis, Vereinigung der Württembergischen Augenärzte, June 7, 1914, *Klin. Monatsbl.*, 1914, vol. liii, p. 434.
15. Kostenitsch: Ueber einen Fall von Scleritis, *Archiv. f. Augenheilk.*, 1894, vol. xxviii, p. 27.
16. Parsons: Annular Scleritis, *Ophthalmic Review*, 1902, vol. xxi, p. 181.
17. Parsons: *Trans. Ophthal. Soc. of U. K.*, 1903, xxiii.
18. Saemisch: Fibrom der Sklera, *Archiv. f. Augen- und Ohrenheilk.*, 1872, vol. ii, part 2, p. 115.

SALVARSAN IN INTERSTITIAL KERATITIS AND IN OPTIC NERVE LESIONS.

ARNOLD KNAPP, M.D.,
New York City.

To determine the value of salvarsan in ocular therapeutics, it seemed essential to subject those diseases to treatment in which the old method of treatment had not been successful. This paper, therefore, is limited to a consideration of salvarsan in interstitial keratitis and in optic nerve diseases.

Salvarsan at present is generally given in small doses, frequently repeated, combined with Hg and KI. During the past two years we have taken cases of interstitial keratitis into the hospital and have followed a uniform course of treatment. These patients were given Hg by inunction once or twice daily, small doses of KI, and neosalvarsan in five one-half doses at three days' intervals; then after two weeks salvarsan was given in one-half doses every three days

for five times. The Hg and KI were then continued. The question of further salvarsan was made dependent on the clinical course: it has not been found necessary to repeat it. The Wassermann test did not help us any in determining the effect of the treatment, as in all of these cases it remained strongly positive, as has been the experience in all laboratories.

Of eleven cases, the process remained localized to one eye during the period of observation in six. The resultant vision was $\frac{20}{20}$ in one, $\frac{20}{30}$ in two, $\frac{20}{40}$ in two, and $\frac{20}{50}$ in one. Notwithstanding treatment, the other eye became affected in one; the ultimate vision was $\frac{20}{30}$ and $\frac{20}{40}$.

In a group of relapsing interstitial keratitis there was no improvement in one, and visual improvement to $\frac{20}{50}$ in another. In two cases which had been going on without treatment for four to six months there was partial visual improvement: $\frac{20}{50}$ and $\frac{20}{100}$ in one; $\frac{20}{50}$ and $\frac{20}{70}$ in the other.

We have the impression that the earlier the treatment is begun, the better are the results obtained. In all cases the inflammatory symptoms are quickly overcome. Salvarsan seems to act best in cases with inflammatory symptoms; its action is slight in cases of corneal infiltration without vascularization. The majority of the cases seem to do much better than with the old treatment. Great improvement in the general health was very noticeable. There are, however, cases which are not benefitted beyond the relief of the inflammatory symptoms and in which the corneal infiltrations do not change. Salvarsan has not been able to prevent the other eye from becoming affected. While the expectation on salvarsan in interstitial keratitis may have been placed too high, I do not share the belief, which many hold, that it is of no value in this disease. The results in the majority of cases, in my experience, if the treatment is persevered in, are very encouraging.

The optic nerve lesions are a somewhat more important

field, as they lead to blindness unless the process is arrested. These lesions comprise optic neuritis due to lues and the optic atrophy associated with tabes and paresis. Our knowledge of the primary causes underlying these conditions has been greatly advanced in recent years. An examination of the cerebrospinal fluid permits a rapid orientation of the condition of the central nervous system. In paresis and tabes W. Boyd states that the presence of a lymphocytosis and a positive Wassermann reaction in the cerebrospinal fluid is worth all of the other physical signs put together. The anatomic investigations of Stargart in tabes and paresis have shown that the optic atrophy is not due to an ascending degeneration starting in the ganglion-cells of the retina, but is caused by a lesion situated generally in the intracranial part of the optic nerve and in the chiasm. In this lesion degenerative processes are always associated with exudative ones, the latter preceding the former. The exudative processes resemble the changes in the cerebral cortex in paresis and about the posterior nerve-roots of the cord in tabes. A further confirmation of this is the demonstration by Noguchi of the spirochæte in the tissues of the central nervous system.

The opinion which was formerly expressed that salvarsan exerted an unusually deleterious action on the optic nerve, producing a rapid blindness with atrophy, has apparently not been confirmed. Experimental work and also clinical experiences have shown that salvarsan does not exert any unfavorable action on the optic nerve nor on any other part of the eye. Unpleasant reactions, the so-called Herxheimer reaction on the part of the optic nerve, can be avoided by beginning with very small doses of neosalvarsan.

In our series there are two cases of optic neuritis with central scotoma. Both were obstinate cases which had not been improved with Hg and KI. Tentative initial doses of neosalvarsan, 0.35 gm., were begun with, and the strength slowly increased. Mercurial inunctions and KI were ad-

ministered at the same time. In both cases the vision improved and the field enlarged.

It is very much more difficult to decide upon the value of treatment in optic atrophy. There can be no question but that our results in this affection by the older methods of treatment are absolutely negative. A number of features, however, suggest that the outlook for a more successful treatment is of promise.

The choroid plexuses are said to be impermeable to foreign substances when in a normal condition. Hence remedial agents introduced into the body in the usual way do not gain access to the cerebrospinal fluid in a sufficient degree to fulfil the requirement that the organism and the agent be brought in close contact. An attempt to overcome this is furnished by the intraspinous treatment with salvarsanized serum, as suggested by Swift and Ellis. This method of treatment has been carried out in our cases by Dr. George Draper. These cases, in brief, are as follows:

E. C., male, aged fifty-two. Diplopia, pupils irregular and fixed. Vision reduced to $\frac{1}{200}$. Very contracted field. Optic atrophy. Has been treated with Hg injections and salvarsan without avail. Spinal fluid showed Wassermann + ; cell count 75; globulin +. After intraspinous treatment the cell count was reduced to 13. No improvement in eyes.

O. B. P., male, aged fifty. Optic atrophy. No vision. Spinal fluid: cell count 133; globulin ++; Wassermann +. After repeated intraspinous injections cell count 7.5; globulin 0. Eyes, no improvement.

F. G., male, aged thirty-nine. Diplopia for nine years. Pupils medium; Argyll-Robertson. V. = $\frac{20}{20}$ each. Optic discs pale. Field shows color contraction. Wassermann: blood +; liquor, faintly + ; cell count 47; globulin +. Repeated intravenous and intraspinous treatment gave cell count 6; globulin + −; Wassermann −. No particular change in eyes.

H. K., female, aged forty-eight. Optic atrophy. V. R. E. = $\frac{2}{70}$; L. E. = l. p. Field, right, concentric contraction. Pupils immobile. Wassermann: blood and liquor strongly +; cell count 85; globulin +. After four intravenous salvarsan and one intraspinous cell count reduced to 11. No change in eye condition.

J. W., male, aged thirty-eight. Pupils irregular and fixed. V. = $\frac{2}{50}$. Odd: left, pale; right, n. Fields, some contraction for colors. Wassermann: blood −; liquor, faint +; cell count 53; globulin +. After Hg and intravenous salvarsan the cell count was reduced to 15. There was an actual clinical improvement. The color of the left disc became more pink, the color field improved, and the pupil became more active. After six months the condition had again relapsed, and the cell count was 52. Two intraspinous and general treatments brought about the previous improved state.

Of these five cases, four were not improved, one was improved, and it would have been strange if the outcome were otherwise, in view of the pathology of the lesions.

I fully realize that the number of observations here presented are too few to permit definite conclusions; at the same time, they suggest the following:

There is some question how much salvarsan given intravenously reaches the cerebrospinal fluid when the choroid plexuses are pathologically changed; if they are normal, the amount is infinitesimal. In any case intravenous treatment should first be tried and its effect on the liquor studied. If the abnormal conditions in the liquor are not corrected, salvarsan should be given intraspinally until the cerebrospinal fluid is normal and stays normal on repeated examinations. It has been shown experimentally that staining fluids injected intraspinally reach all parts of the central nervous system, even the perineural sheaths of the olfactory filaments. It is therefore probable that salvarsan reaches the peripheric parts of the optic nerves.

A point to which Draper particularly refers is the importance of the localization of the pathologic process in cerebrospinal syphilis, just as it is in anterior poliomyelitis. The uselessness of endeavoring to restore a degenerated neuron by any known agent must be self-evident. If the degeneration depends on an exudative process due to the direct action of the spirochæte, our aim should be the preventive treatment of these lesions. This calls for the proper examination of the cerebrospinal fluid and its appropriate treatment long before degenerative lesions come on. In this, salvarsan is a distinct accession to our older and still indispensable forms of treatment with Hg and KI.

In other words, the treatment of tabic atrophy is a preventive one. When the cases with visual symptoms come to the oculist it is probably too late. On the other hand, cases of ocular muscle paralysis or with pupillary symptoms, where the optic nerves are still unaffected, furnish a more hopeful field for therapeutic endeavors.

DISCUSSION.

DR. JAMES BORDLEY, JR., Baltimore, Md.: During the past two years I have had all my cases with supposed syphilitic eye lesions examined intraspinously. In 75 per cent. of the cases in which the blood Wassermann was negative the spinal Wassermann was positive. I have seen treated with salvarsan about 200 cases with eye lesions, and my own impression is that it is of doubtful value. The only cases that I have seen in which brilliant results have been obtained were those with exudative lesions in the choroid. These results usually come at a time when you have about given up hope. I have had four cases with atrophy of the optic nerve treated intraspinously. Unfortunately, I was able to get the full details of only two of these cases, and I really only watched their progress, as they were referred to Dr. Albert Keidel and treated by him.

The first was a case of atrophy secondary to locomotor ataxia (Fig. 1). The principal symptoms were: Argyll-

Robertson pupil, atrophy of the optic nerves, loss of patellar reflexes, and positive Romberg. The blood Wassermann was negative, spinal Wassermann positive. A positive history of syphilis fifteen years ago.

In all, four doses of salvarsanized serum were given intraspinously. There was no improvement in vision; in fact, there was a rapid decline coincident with its use. After a

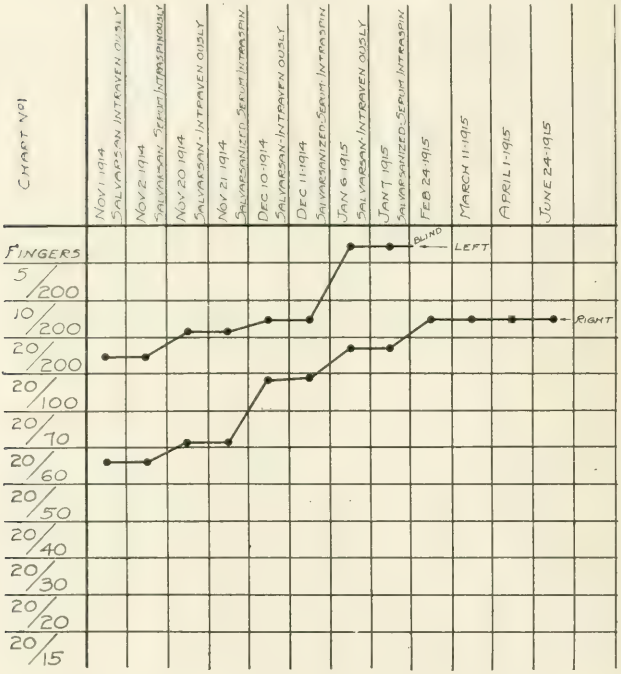


Fig. 1.—Case 1.—Effect of treatment on vision.

lapse of several months the left eye became blind. The treatment was started in November, and by January there was a marked loss in the fields of vision. In April, however, with the central vision showing no improvement, the field began to broaden out (Fig. 2), and each month since there has been a change for the better in the fields, but not in central vision.

The second case (Fig. 3) was one of progressive paralysis secondary to a syphilitic infection. His principal symptoms were: Argyll-Robertson pupil, syllabic speech, irregular handwriting, moroseness, and atrophy—complete, of the right optic nerve; incomplete, of the left. Deep reflexes increased, motor strength diminished.

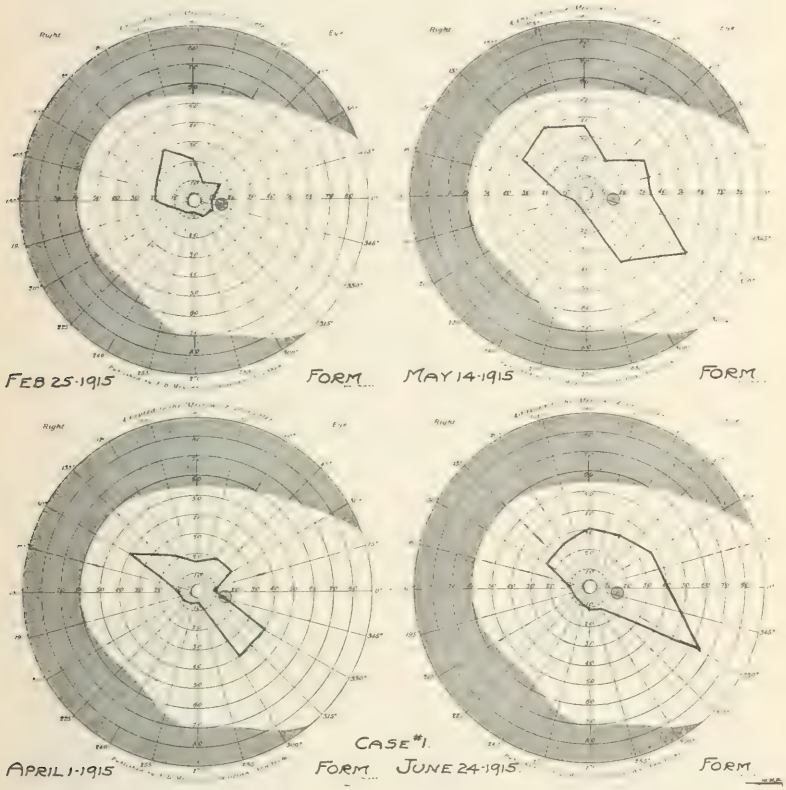


Fig. 2.—Case 1.—Visual fields during the treatment.

The blood Wassermann was negative. The spinal Wassermann was positive, gold chlorid +, globulin +, cell count 36.

During the whole treatment his spinal Wassermann remained positive, but there was a marked diminution in the

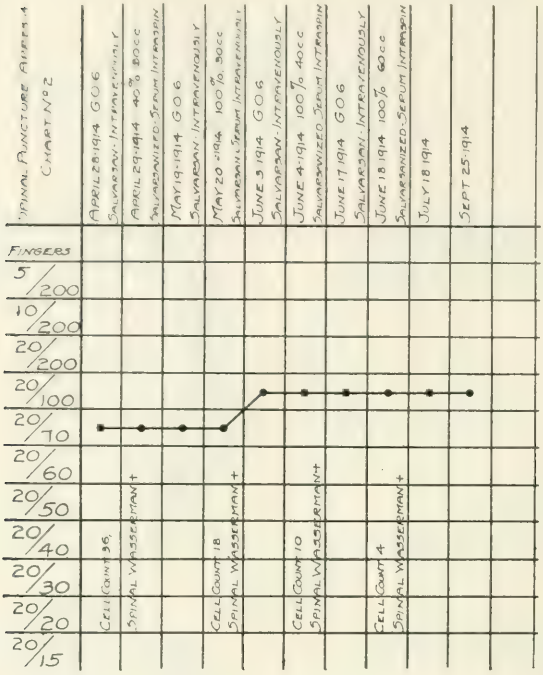


Fig. 3.—Case 2.—Effect of treatment on vision.

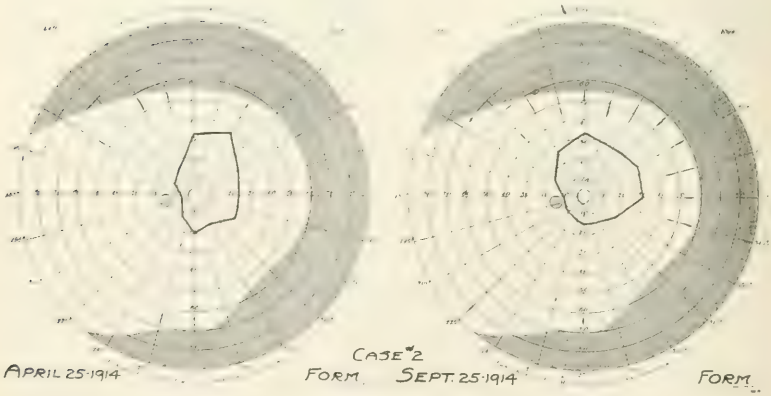


Fig. 4.—Case 2.—Visual fields during treatment.

cell count. His central vision declined, and his fields (Fig. 4) remained about the same. Subjectively he was not bettered by the treatment; in fact, his son wrote me that he was quite sure he was failing mentally and physically.

DR. KOLLER: I believe it erroneous to speak of the lack of danger in the Herxheimer reaction and cite the following example. I have now under my care the child of a syphilitic father who went to Aix-la-Chapelle to be treated for syphilis. His children were all subjected to the Wassermann test, although they were in good health and gave no clinical evidence of the disease. The reaction was positive, and, owing to the zeal of some physician, they received salvarsan. Following this treatment, one of them, a young girl of seventeen, had an outbreak of interstitial keratitis. It was one of the worst cases I have seen, has lasted two years, and has not cleared up yet. I wish to draw attention to this danger of salvarsan treatment when given to persons who have no symptoms of syphilis, but show a positive Wassermann reaction.

DR. PETER A. CALLAN, New York: Dr. Knapp appears to have had very satisfactory results from the use of salvarsan. In cases of lesions of the optic nerve I have seen what I consider disastrous results from its use. Regarding interstitial keratitis, he seems to think that salvarsan has prevented the development of the disease in the second eye. We know that in the acquired form of syphilis with interstitial keratitis it is unusual for the disease to attack more than one eye, although in the inherited form the second eye is invariably attacked. The intervals between the attacks are very long—five, ten, or even sixteen years. If, however, the patients do not seek treatment inside of a month, both eyes are usually affected. Therefore, I hardly think that there has yet been time to feel sure that the disease is not going to affect the other eye in Dr. Knapp's cases. I have always seen this occur in the inherited form, but it is the exception in the acquired form to have it affect the second eye.

DR. W. B. MARPLE, New York: It seems to me that the treatment of diseases of the optic nerve by salvarsan bids

fair to change entirely the prognosis of these conditions. We all remember what a hopeless outlook, until recently, confronted the unfortunate patient with optic-nerve atrophy. I have the records of some seven patients with this disease who have been under my care during the last year, and who have been treated by intravenous and intraspinal injections of salvarsan. This does not include several others who have been treated at the hospital, as it has not been possible to follow these cases. I am quite sure that, had I seen these cases five or ten years ago, most of them would have been blind by this time. The first patient was seen May 14, 1914. Both nerves very pale; no perception of light in the left eye; corrected vision, right eye, $\frac{2}{20}$; field shows a quadrant defect in the lower nasal portion. He has had five intravenous injections of salvarsan and five intraspinal injections of serum to which .1/4 mgm. of salvarsan had been added, these all given by Dr. Fordyce. The serologic findings in four months were greatly improved (spinal Wassermann becoming negative), as well as his general condition. May 28, 1915, vision in the right eye was the same, as was also the right visual field.

Another patient, seen first November 14th, seemed quite hopeless. He had perception of light to the temporal side in the right eye, and fingers at three feet in the left. Four months later the vision of left eye had increased to $\frac{10}{200}$. During this period intravenous and intraspinal injections of salvarsan were given by Dr. Baketel. A third patient—and I am indebted to Dr. de Schweinitz for the opportunity of watching this case—had marked optic-nerve atrophy in both eyes, following specific infection in 1903. November, 1914, V. = R. E., $\frac{3}{200}$; L. E., $\frac{20}{100}$. During the next five months he had six intravenous and six intraspinal injections of salvarsan by Dr. Fordyce, followed by a great improvement in the serologic findings and in his general condition. April 17, 1915, vision of the right eye had increased from $\frac{3}{200}$ to $\frac{10}{200}$. In the left eye unaided it was still $\frac{20}{100}$, but the correction of some astigmatism, which I regret I had not previously attempted, brought his left vision to $\frac{20}{30}$, and with the correcting glass he could read his music (he was an organist). I cannot say how much of this improvement was due to the glass and how much to the treatment. I do know, how-

ever, that his vision with correction increased from $\frac{2}{4}\frac{0}{0}$ in January to $\frac{2}{3}\frac{0}{0}$ + in April, so that, counting on a similar increase from November to January, the vision at the start with correction could not have been more than $\frac{2}{7}\frac{0}{0}$, if that.

Another case has been under observation for some months. After going two years or more with good vision following a combined extraction, he had an iridocyclitis with vitreous full of opacities. After running a slow course for several months the eye quieted down, the opacities began to clear away, and for a while there was a gradual visual improvement. This did not continue with the progressive clearing of the vitreous, and I found the optic nerve pale, and the visual field concentrically contracted. He was then examined by one of our most skilful men, who, though from his other symptoms was convinced that the patient had lues, failed to get a positive Wassermann. As we know, these cases are sometimes given a provocative dose of salvarsan, when the reaction then becomes positive. The consultant, however, said that this might be very dangerous in such a case on account of the so-called Jarisch-Herxheimer reaction. Even before the days of salvarsan it was observed that the macular and papular eruptions, after the administration of mercury intramuscularly or after inunctions, sometimes became much more intensely red and small. In explaining this phenomenon they assumed that the spirochætæ were killed in large numbers and their endotoxins released. The local intensity of the syphilitic process they thought was due to the action of these endotoxins. Since the advent of salvarsan the Herxheimer reaction in the early eruptions has been noted much more frequently, for the reason that this drug has a much quicker effect in killing spirochætæ than mercury. An increase in the intensity of the process has also been noted in the central nervous system and about the cranial nerves. A small injection of salvarsan apparently activates or intensifies the local condition, which, however, as a rule yields to persistent treatment. Hence the consultant wisely decided to put the patient on small doses of mercury for a while, rather than to risk a provocative dose of salvarsan.

I desire to emphasize one point, and that is that in our enthusiasm over the newer treatment we should not forget the old. Much better results will be secured if those who

are carrying out the newer treatment of these cases work hand in hand with the oculist, who oftentimes has a larger experience in the older methods of treatment. The importance of this point is shown in a case of ophthalmoplegia interna of the right eye which I first saw in November, 1914. He was treated by one of our most skilful men, with a great improvement in the serologic findings but little improvement in the ophthalmoplegia. I then found out that he had not been getting any iodids. When we treated this class of cases ourselves I almost invariably cured nuclear paralysis by large doses of the iodids. This patient is now taking large doses of this drug.

Of course, these cases have not been under observation very long, and we must not be too sanguine. Time alone will tell; but, so far as we have been able to observe them, this method of treatment bids fair to save the vision of many cases which we previously regarded as hopeless.

DR. R. L. RANDOLPH, Baltimore, Md.: At the last meeting of this Society in Washington this question was discussed, and I recall making some observations on the difference in the results from the use of salvarsan in cases of interstitial keratitis of the hereditary and of the acquired form. My later experience has been entirely confirmatory of the observations I made then. That is to say, in interstitial keratitis due to inherited lues I got no results from the use of salvarsan, but from its use in the acquired form the results have been brilliant, just as Dr. Callan has pointed out. I think that probably the location of the organisms of lues has something to do with the results in these cases. In the inherited form of the disease the spirochætæ are only exceptionally found in the cornea or the aqueous, but in the acquired form they are frequently found in these places. That probably accounts for the good results. In the cases of acquired lues which I recall potassium iodid and injections of mercury salicylate were used; but after the use of salvarsan I was struck with the rapidity of the disappearance of the clouding of the cornea and of the disturbance of vision.

DR. GEORGE S. DERBY, Boston, Mass.: I should like to say a few words about the systemic treatment of interstitial keratitis. Two years ago, before this Society, I urged that

cases of interstitial keratitis be looked on not merely as patients afflicted with a disease of the eyes, but rather as victims of a dangerous general disease, of which the ocular condition is but a single phase. Very few ophthalmic clinics are so organized that it is possible to follow these unfortunate children and young adults to the period when it is safe to discharge them from treatment, for antisyphilitic treatment should by no means cease when the ocular disease is at an end, yet this is what happens in most cases of interstitial keratitis. We lose track of them, and then, perhaps in a year or two, they turn up at a surgical or orthopedic clinic with painful, swollen knees. Next we may see them in the aural clinic, and so on. I do not think that it can yet be said with certainty whether any of the complications of hereditary syphilis can be avoided by an early and persistent course of treatment, or whether or not the severity of these complications can be influenced by such treatment. It seems reasonable, however, to suppose this to be the case; and Dr. Knapp's admirable paper points toward it. I believe that we have a duty toward these patients, beyond the treatment of their eyes, to see that each one of them is placed in competent hands for the treatment of his hereditary syphilis. I take it that few of us are experts in the treatment of this disease, and expert treatment is none too good for these unfortunate people.

Four or five years ago such a system was instituted in Boston. From the Carney Hospital, and from certain of the clinics at the Massachusetts Charitable Eye and Ear Infirmary, cases of interstitial keratitis were sent to the syphilis clinic at the Boston Dispensary, presided over by Drs. Abner Post and C. Morton Smith. At present a large proportion of all cases of interstitial keratitis coming to the Infirmary are handled in this way. Under this system the eyes are regarded as but an incident in the course of the disease. Moreover, Drs. Post and Smith do not stop there. Each one of these patients means a syphilitic family; and, with an exceptionally efficient social service organization, it is possible for them to reach out and gather in other members of these families. I have been much impressed with the amount of good done to these children by long-continued treatment. In nearly all instances there is a noticeable improvement in

the general condition of the child; so that, from being a backward, sickly youngster, he becomes strong and vigorous and mentally much more alert. Through this system we are now getting some figures about the cases which should prove valuable. Under the leadership of Dr. Post an investigation of 100 cases of healed interstitial keratitis is in progress. A grant of money for this purpose has been obtained from the Perkins Institution for the Blind.

In view of Dr. Knapp's excellent results, it seemed of interest to inquire how good vision was obtained among this group of cases. Out of 63 cases already examined, I find that 23 had one eye with a vision of $\frac{2}{3}\frac{0}{0}$ or better, and seventeen of these had $\frac{2}{3}\frac{0}{0}$ in one eye. These 23 cases represented 44 eyes; and of these, 23 had a vision of $\frac{2}{2}\frac{0}{0}$, eight, $\frac{2}{3}\frac{0}{0}$, and five, $\frac{2}{2}\frac{0}{0}$ in each eye. In just what degree salvarsan was responsible for these good results I am unable to say as yet.

There is one thing to add. I believe that the time is coming when we shall have a better chance to treat hereditary syphilis before destructive lesions have made their appearance. To do this the disease must be recognized at an early date—in infancy. Only a comparatively small percentage of these infants showed active, recognizable signs of the disease in very early life, except, perhaps, to the expert. A start might be made in our maternity hospitals. It is not practicable to test the baby's blood; for, in the first place, it is a somewhat difficult procedure to obtain it. In the second place, unless active signs of the disease are present, the reaction is apt to be negative. Why, however, should not each mother's blood be tested, or perhaps the placenta might be used for the purpose? Given a positive reaction, it would be logical and justifiable to label that child as a suspect and treat it and the mother accordingly. If this were done, and I am confident that at some future time something of the sort will be done, a long step forward in the control of the disease would be taken.

DR. JOHN R. SHANNON, New York: With reference to the Herxheimer reaction in interstitial keratitis, I would like to mention a case which I had during the past winter in a girl of eighteen years, who had a violent attack of interstitial keratitis in both eyes. She was given mercurial inunctions,

as well as the usual treatment for the eyes themselves. The disease appeared in both at the same time. In three weeks an intravenous injection of salvarsan was given. The disease, in spite of this treatment, pursued a virulent course. Both corneæ were densely opaque, and there was deep ciliary injection, with photophobia, while her general condition suffered seriously. Within three weeks after the injection of salvarsan the appearance of the corneæ changed slightly, and instead of being grayish-white they became yellowish. I brought the girl into the hospital. The epithelium on the surface of both corneæ broke down, and she proceeded to have a necrosis of the corneal tissue. There was a dislocation of the lens of the left eye into the anterior chamber. The process was not so severe in the right eye, and the lens in that eye remained in place. By means of a pressure bandage applied between the necessary treatments the corneæ were prevented from rupturing, and the result was that she has now no vision in the left eye, in which the dislocation of the lens occurred, and $\frac{5}{200}$ in the right eye, the cornea of which has cleared up considerably. The question is whether this was an instance of the Herxheimer reaction or not.

DR. E. TERRY SMITH, Hartford, Conn.: During the past few years we have used salvarsan in many acute and chronic luetic conditions of the eye. It has apparently been of considerable benefit in acute cases. The chronic cases, as a rule, have not seemed to be improved.

About eight months ago, at the suggestion of Dr. Stoll, of Hartford, I started using serosalvarsan. It is prepared by drawing the blood one hour after an intravenous injection of salvarsan; the blood is then centrifuged for one hour, and is then inactivated for half an hour at 56° C. The serum may then be put in glass tubes and sealed up. If this is done, it will keep for a considerable period of time.

At first we used the serum as an eye-drop, dropping one drop in the eye every hour during the day. In some cases it was used full strength and in others diluted by saline solution. Of late we have been following the technic suggested by Dr. Lamb, of Washington, *i. e.*, injecting 1 c.c. of serum under the conjunctiva in the lower quadrant of the eye. The

day after the injection of salvarsan the eye is dusted with dionin. This treatment should be carried out twice a week unless the reaction is too great.

I feel quite certain that in serosalvarsan we have an aid in treating superficial, acute and chronic syphilitic conditions of the eye. The serosalvarsan is always to be used in conjunction with proper constitutional treatment, as in almost all cases the eye alone is but one manifestation of systemic infection.

DR. T. B. HOLLOWAY, Philadelphia: I have not seen a great many cases of interstitial keratitis in which salvarsan has been used, but in those instances where it was used the results were gratifying, but I believe repeated injections to be necessary. I recall a case of acquired luetic involvement of the cornea that occurred in the Eastern Penitentiary, in which 13 injections were required before much effect was obtained. Then the result was most satisfactory, not only as to the integrity of the cornea, but for vision. I know of at least four cases of advanced optic atrophy where, after an injection of salvarsan, the patient stated the vision had promptly become nil. During the past two years I have observed two cases in which optic neuritis or neuroretinitis developed within a month after a negative Wassermann reaction. In the first patient I was uncertain whether this condition had developed as the result of previous injections or whether it was dependent upon the disease itself, but in both instances the lesion subsided, with favorable results after subsequent salvarsan injections.

In view of Dr. Bordley's suggestion, I may state that I think it probable that had spinal fluid been used instead of blood, a positive reaction would have been obtained, as in both instances the ocular affections were undoubtedly specific.

DR. S. LEWIS ZIEGLER, Philadelphia: I have previously reported some cases of interstitial keratitis in which the use of salvarsan has brought about excellent results, but I have not used it in optic-nerve lesions, owing to the earlier statement that there was danger from that course. About 17 cases of paralysis of the aural nerve were reported as occurring in Vienna following the use of salvarsan. I discussed

this question with Dr. Ehrlich himself, and he suggested the idea that the first injection had set free a quantity of toxins which had injured the nerve; but he thought that, if within a week the injection had been repeated, these toxins would have been dissipated and prompt recovery from the nerve lesion would have followed.

While the injection of salvarsan has usually proved successful, it has not always done so. Not long ago I showed a patient before the Wills Hospital Ophthalmic Society in which salvarsan had been used, in the service of one of my colleagues, without success. In this case I used an intravenous injection of enesol, a preparation which Dr. Darier, of Paris, uses constantly in his clinic. The enesol, which is a solution of the salicylates of arsenic and mercury, brought about great improvement in the patient's condition. I substituted enesol for salvarsan for two reasons: one, that salvarsan had previously failed to cause improvement, and the other that salvarsan was difficult to obtain at that time on account of the war. Enesol should be given a fair trial, for, although it is new, it has proved itself most efficient. No treatment will always be successful in any series of cases. I have always insisted on continuing the use of mercurial inunctions in the intervals between injections, as was recommended by one of the speakers to-day.

DR. W. H. WILDER, Chicago, Ill.: When we see a case of interstitial keratitis from congenital syphilis we expect to have it under treatment for a period of at least seven or nine months, and generally for a much longer time. Before the use of salvarsan I cannot recall a case of this kind in which the inflammation seemed to run its course in less than five months, and such a short period is a rare exception. Now, if by any means this period is materially shortened, it is only fair to assume that it is because of the treatment. With the usual antisyphilitic measures the course of the disease, in most cases, does not seem to be markedly influenced, and even while treatment is being vigorously practised, the disease will sometimes begin in the other eye. I have had three cases in which the duration of the disease was so distinctly shortened by the use of salvarsan that I must attribute it to this new method of treatment.

The first was a child of five years, with well-marked syphilitic signs, whose mother gave a history of syphilis. Wassermann test of the child was positive. Both eyes had been affected four weeks when I first saw the case, the corneæ were extremely infiltrated, and there was new vessel formation. Three injections of neosalvarsan were given intravenously, and within six weeks the corneæ had cleared and the eyes were to all appearances well.

In a second case, a girl of about twelve years, one eye only was affected, and was seen soon after the beginning of the disease. She received six doses of neosalvarsan intravenously, and in two months the cornea began to clear and all sign of active inflammation of the eye disappeared. The other eye did not become affected while she was under observation.

In a third case,—a recent one, a boy of five years,—marked interstitial keratitis affecting both eyes had been present for several weeks before the treatment with salvarsan was started. In this case seven or eight intravenous injections of neosalvarsan were given, and it was three months before the active signs of the disease subsided. There is now, about six months after I first saw him, considerable cloudiness of each cornea which is rapidly disappearing.

It seems to me that if the case can be treated in this manner early enough, before the exudate in the cornea becomes organized, there is a much greater chance of securing a clear cornea. In connection with this subject of the use of salvarsan in the treatment of interstitial keratitis it seems proper to refer to a recent article by Seibert, of Washington, on Subconjunctival Injections of Salvarsanized Serum in Ocular Syphilis that appeared in the *Journal of the American Medical Association*, May 15, 1915.

The method of treating syphilis of nerve tissues by injection of salvarsanized serum into the spinal canal suggested to him the treatment of syphilitic ocular lesions in a similar manner. He made a report of several cases treated by subconjunctival injections of salvarsanized serum with marked benefit, and following this Lamb made use of the same method in a number of cases, including iritis and interstitial keratitis.

Recently I have used this method in one of the most severe cases of syphilitic iritis I have ever seen. The patient,

a man of about thirty years, had an initial lesion about two years before the outbreak of the iritis. Antisyphilitic treatment for three weeks was apparently without any effect; the iris was firmly adherent to the lens capsule; there was marked hypopyon, and vision was reduced so that he could only distinguish hand movements. Wassermann was strongly positive. He was given a dose of neosalvarsan, four ounces of blood were withdrawn, and serum prepared. Four doses of the salvarsanized serum were injected beneath the conjunctiva at intervals of two days, about ten minims at each dose. The result was surprising. Improvement began after the first dose, and at the end of ten days the vision had risen to $\frac{20}{40}$, and he was allowed to leave the hospital. When last seen, he had recovered from the inflammation, but some synechiæ remained. The result was so gratifying that I shall certainly try the same method in other cases of ocular syphilis.

DR. G. E. DE SCHWEINITZ, Philadelphia: I would like to repeat the experience of my service in the University Hospital with respect to the treatment of interstitial keratitis with neosalvarsan, already reported to this Society two years ago. This experience is in exact accord with that which has been recorded by Dr. Knapp. This treatment shortens the duration of the disease and lessens its activity. All the cases of interstitial keratitis were watched in the hospital, and were treated in conjunction with the genito-urinary department. I am unprepared to maintain that this method of treatment facilitates the disappearance of the exudations or interstitial deposits in the cornea or not, but I believe that it does. I have never seen an evil result from its use. I have seen a few cases in which the treatment was not superior to the old-fashioned treatment. I prefer mercury by inunctions and potassium iodid. My colleagues of the genito-urinary department prefer mixed treatment, *i. e.*, potassium iodid and mercury bichlorid administered in compound tincture of gentian. These remedies are given in the intervals between the neosalvarsan injections.

DR. ARNOLD KNAPP, New York: I have nothing to add, except to reply to Dr. Randolph's remarks by stating that

we have always given ten injections. I do not think that anything can be accomplished with one or two injections. After one or two injections are given with no particular result, the doctor is apt to lose faith in the method of treatment and discontinues it.

Regarding the treatment of optic atrophy, I think that this is a very doubtful field for this method. What I wanted to suggest was that we as oculists have an opportunity, in the presence of pupillary changes or of ocular muscle paralysis of the pre-optic-atrophy stage, for a thorough examination of the spinal fluid; treatment then directed toward making the cerebrospinal fluid as nearly normal as possible offers some hope of preventing the optic atrophy.

EXHIBITION OF NEW INSTRUMENTS AND APPARATUS.

THE IVES-LANGDON PHOTOMETER.

H. MAXWELL LANGDON, M.D.,
Philadelphia.

While there are but few cases in which a study of the light sense is required in order to arrive at a proper diagnosis of the condition, and while the study of this function cannot in any way compete either in importance or interest with the study of the senses of form or color, yet it seems a subject which is worthy of something better than the position of complete neglect, which, to judge from present-day ophthalmic literature, it now occupies.

This lack of interest is probably due to three factors: First, the above-mentioned needlessness of studying this function in order to arrive at a correct clinical diagnosis; second, the time required in making the retinal adaptation, the average office not offering many absolutely dark corners where a patient may be placed for a space of time bordering

on a half-hour without so interrupting the day's work as to make such an examination, at least during office hours, well-nigh impossible; and, third, the well-recognized fact that there is no piece of apparatus so arranged that it is easy to manage and so standardized in construction that two or more observers working with different instruments may expect to arrive at approximately similar conclusions. To the first two reasons suggested for lack of interest in the study of the light sense there is but little to say: the first cannot be controverted, but the second may be met with a bandage sufficiently thick as to effectively prevent all rays of light from reaching the retinas. It is to supply the lack of a standardized apparatus that Dr. Herbert E. Ives and I have devised and had made the one which is here presented for your consideration, but before going into the details of its construction a short review of previously used instruments and the reasons for their lack of accuracy would seem advisable.

The reason for the rejection of Chibret and Izard's contrivance is not far to seek when it is remembered that the light source used by them was the sky; one with greater variability or less under control could hardly be conceived. Forster's photometer, the one undoubtedly most commonly used, with the possible exception of Bjerrum's or similar charts, suffers from three defects: First, the source of light is a standard candle, which varies greatly in illuminating power with variations in atmospheric pressure, condition of wick, etc.; second, the light is diffused through oiled paper, introducing a second variable; and third, it is reflected to the eye under examination and the gage read when certain objects become visible, introducing the sense of form. The above-mentioned charts of Bjerrum, Landolt, and others also introduce an element of the sense of form, and so are not well adapted to study the light sense only.

In 1896 R. Wallace Henry made a great advance when he

had the eye view an aperture illuminated by transmitted light, filtered through variable thicknesses of opal glass, but he still used the standard candle as his light source, as did Percival J. Hay in 1905, who, however, improved on Henry's apparatus by having the intensity of the light altered by polarization by means of Nicol prisms. His seems to have been the first instrument so devised that the light minimum and the light difference could be studied at one sitting.

Nagel in his "adaptometer" uses three osmium lamps as the light source, varying the amount of light by an Aubert slide adjustable from 1:10,000 square millimeters and by three screens, each with a darkening power of twenty. He reverses the usual procedure in making his observation, instead of having the retina to be tested adapted in the dark, he exposes it to a midday or bright artificial light for fifteen or twenty minutes and then measures the adaptation time for different amounts of light. The amount of light required to produce sensation varies, according to Nagel, from 1 meter candle, immediately after adaptation, to $\frac{1}{270,000}$, after sixteen hours in the dark. Nagel, in using the meter candle, has the most accurate gage of surface brilliancy, and this we have kept in our contrivance, though for clinical purposes a less complex scale of measurement is offered. We felt the two things most desirable were absolute steadiness of the light source, and a method of gradually and imperceptibly changing the amount thrown on the retina. Electricity with a milliameter and rheostat in the circuit was decided on as the best means of securing the former, and an opening altered by an iris diaphragm seemed to give us the control we desired.

We have taken the Henry apparatus, which consists of a box 33 cm. long and 16 cm. square, one end open and covered with a large square of black velvet attached to the top and sides, the inside of the box being black, and the other end having a two-millimeter opening, the spot to be illuminated.

To this we have added a somewhat similar box, 27 cm. long and 17 cm. square, divided into two portions,—the diffusion chamber, 17 cm. square, lined throughout with porcelain, 2 mm. thick; and the light chamber, containing the lamp, 10 cm. long and 17 cm. square,—these being separated by a partition, in the center of which is inserted the iris diaphragm with an opening variable between 6 mm. and 33 mm. and controlled by a lever, connected on the outside with a milli-

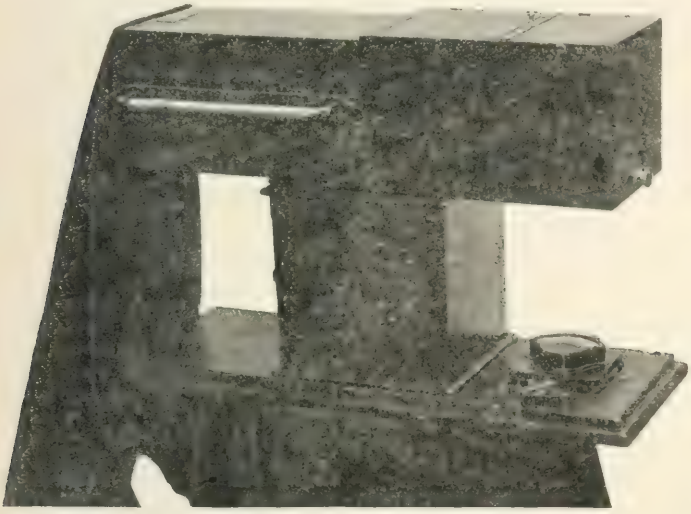


Fig. 1.—The Ives-Langdon photometer.

meter scale. The lamp in the light-chamber is a standard incandescent, tested and sealed, and guaranteed to burn without change for one hundred hours. It is fixed against the distal wall of the chamber, opposite the opening of the iris diaphragm. It was our belief that one thickness of the 2 mm. thick porcelain would be sufficient to dim the light before it entered the diffusion chamber, but we had to place two more pieces in the opening, making the light pass through 6 mm. of porcelain before it falls on the sight-hole.

The proceeding I use is as follows: The adaptation time is twenty minutes, this time being chosen since it has been shown that the curve of adaptation rises rapidly the first twenty minutes, slightly the next ten, and after that so slowly as to be negligible except for most exhaustive tests. The patient is then placed with the head covered by the black velvet hood, one eye being occluded, and he is instructed to fix the center of the instrument, both hands resting on its sides, using the muscle sense as a guide. The current used is 0.3 milliampere, and the iris diaphragm is then slowly opened. I have tested the eyes of 50 people, between the ages of ten and fifty years, all the eyes having normal form and color senses, full visual fields, and not more than two diopters of refractive error. The average opening of the iris diaphragm required to allow enough light to pass into the diffusion chamber to illuminate the sight-hole sufficiently to produce retinal stimulation is 15 mm.

Dr. Ives has made a photometric reading of the surface brilliancy of the porcelain plate over the sight-hole, and has calculated the illumination .0013 $\frac{c\,p}{m}$ when the iris diaphragm is this size; knowing this, the surface brilliancy in meter candles can be calculated with any size opening, or, if the observer does not desire to go to this trouble, the record may be made in millimeters of diaphragm opening.

This instrument was made by Wall & Ochs, Philadelphia.

DISCUSSION.

DR. CHARLES H. WILLIAMS, Boston, Mass.: In the measurement of lights, one of the most important factors is the photometer screen—one that will measure the lights accurately and with the smallest amount of fatigue to the eyes of the observer.

The Bunsen screen, first made in 1841, is one of the simplest and most widely used. It consists of a sheet of white paper in which a central spot or figure about an inch in diameter is made more transparent than the rest of the paper

by impregnating the central spot with paraffin. By moving such a disc along a photometer bar a point will be found where the light transmitted through the central spot is about equal to the light reflected from the surrounding paper, so that the spot nearly disappears. In this position the distance of the disc from the lights placed at each end of the photometer bar will give the proportional intensity of the two lights.

A more sensitive and accurate form of screen is the Lummer-Brodhun, which consists of a rather complicated optical train, by means of which light from the two sources to be compared is brought into separate parts of the field as seen through the monocular telescopic sight-tube. When the two lights are equal, the field appears uniformly illuminated, but when they are not equal, there is a contrast in the brightness of the different parts of the field. The box containing the optical train is moved right and left along the photometer bar until the point of uniform illumination of the whole field is reached, and the distances of the two lights from the screen then gives a proportionate measure of their intensity. With this complicated apparatus only one eye can be used at a time; also the determination of the point of uniform illumination of the field soon causes considerable fatigue of the eye, so that with any long series of measurements the ocular fatigue is very uncomfortable.

I have been working for some time to devise a photometric screen which will have some of the simplicity of the Bunsen screen and the ability to make readings quickly, and which will also approximate more nearly to the greater sensitiveness of the Lummer-Brodhun screen.

A sheet of opal glass, about 3 inches square and $\frac{3}{16}$ inch thick, has a round hole $1\frac{1}{4}$ inches in diameter cut in its center; the surfaces of the glass are not polished, but are left fine-ground, and on each side of this plate is another sheet of thin opal glass of the same size ground to a thickness of half a millimeter, with both surfaces fine-ground, not polished. In the center of the round hole in the middle plate of glass is a round button of opal glass half an inch in diameter, with each surface fine-ground, and of such thickness that when the parts are in proper position the thin sheets of glass on each side of the central plate are in con-

tact with the surfaces of the plate and with the surfaces of the central button. This screen is mounted on the photometer bar, and used in the same way as the Bunsen disc, and a number of comparative readings by different observers have shown that it gives mean values more than twice as sensitive as the Bunsen screen, although not so sensitive as the Lummer-Brodhun. It is not trying to the eyes, and can be used as easily and rapidly as the Bunsen screen. The surface of my new screen is uniform; the edge of the button in the center of the opening in the middle plate gives a sharp outline between it and the surrounding thinner portions of the screen, so that a nearly complete disappearance of this central button can be had when the two lights are equal on each side of the screen. Sets of readings by different persons, taking the mean of nine settings, give results which are very close and which come quite near to the readings of the Lummer-Brodhun screen, used with the same lights and conditions.

A Review of the History of the Electric Ophthalmoscope, with a Demonstration of a New Ophthalmoscope, which Consists of a Rearrangement of the Parts of Antecedent Instruments.

J. HERBERT CLAIBORNE, M.D.,
New York City.

I know of no more appropriate way of beginning this paper than by quoting the words of Dr. Dennett in a letter recently addressed to me:

I take the pleasure in writing out for you the history of the electric light ophthalmoscope as it came under my observation.

Some time about the beginning of the year 1884 I took the handle off my Loring ophthalmoscope and replaced it by a hollow pasteboard handle with an electric light inside and a plane mirror on one end. There was no lens in this illuminating apparatus, but it was a very good instrument for

upright image work, and by changing the mirror to a concave it was good for most anything.

About that time I was told that Dr. William Thomson, of Philadelphia, was to exhibit an electric light ophthalmoscope at a meeting of the New York Academy of Medicine. A member of the Academy invited me to be present and to bring my instrument, and in the discussion following the demonstration of Dr. Thomson's instrument I was asked to exhibit mine and did so.

Dr. Thomson's instrument was not a failure, but it was more of a prophecy than a fulfilment, and Dr. Thomson very generously allowed then and there that his was not "in it."

He said complimentary things about mine, and so far as I know never exhibited his again. Shortly after that Meyrowitz made me the instrument which was described in a paper which I read before the American Ophthalmological Society on July 15, 1885. This paper was printed in the Transactions of that year (pages 156 and 157).

In this paper as read and as printed it is stated that the concave lens that was at first put in the handle was unnecessary, and that the proper place for the lamp was in the focus of the condensing lens, with such freedom of motion as to allow of change in the character of the pencil. The Transactions went to press before the illustrated diagrams could be changed, but before the end of the year the condensing lens B was moved up close to the mirror, and no important change has been made in the instrument since. As will be seen from the cut, though badly drawn, the hole in the mirror was made oval (the long and short diameters being related as ten is to seven, for obvious reasons).

Meyrowitz made me a beautiful instrument of this model, and I used it almost exclusively for two years and have never seen a better. There was no demand for it. Electricity was not so widely distributed as a public utility then as now, dry cells were not so much used, and when I came home from the country one fall my equipment was out of order, and between my office and Meyrowitz repair shop the ophthalmoscope got lost and neither Mr. Meyrowitz nor myself ever knew where it went to. As far as I know, this was the only ophthalmoscope with interchangeable handles that was ever made.

I do not know that any other electric light ophthalmoscopes were made immediately after mine fell into disuse, but the description and diagrams were copied in foreign journals (one or more), and in 1900 Dr. Wolf, in the *Berliner klinische Wochenschrift*, changed my model by putting the Rekoss disc before the mirror instead of behind it, and thought he could see the fundus better by so doing. I could see no difference myself except that the dust on the lenses in the disc was sure to be lighted up and made as obtrusive as possible.

I think the above is enough to show that I ought to have the credit of the invention of the electric ophthalmoscope.

Among the surgeons who have modified the instrument are Marple, Harrower, Hare, May, and Koller. In the Transactions of this Society, 1906, Marple suggested the use of a U-shaped mirror with a vertical aperture, and adjusted an arm to the stem of the ophthalmoscope so that the surgeon could move the lamp up and down while using the instrument. This also enabled him to use the indirect as well as the direct method. At first blush it would appear that this was a decided innovation upon the Dennett instrument, but, as Dennett has pointed out, the electric lamp in his instrument is placed in the focus of the condensing lens and can be moved either to or from the lens, so that parallel convergent or divergent rays may be thrown upon the small mirror, thus varying both the intensity of the illumination and the size of the field; likewise, of course, the focus. Marple's mechanical adjustment has rendered this easy and accurate, and has made it possible to use both direct and indirect methods—a thing which certainly had not been done up to that time. Marple, likewise, applied the electric lamp to the Morton ophthalmoscope, but in this latter the illumination was derived from electric cords, controlled through a rheostat, whereas his more recent instrument, just referred to, has its illumination from the dry-cell storage battery carried in the handle. There are one or two other models of electric ophthalmoscopes on the market, illuminated through the cord

system, but I think it safe to say that the advent of the dry-cell storage battery has entirely superseded this, and that all ophthalmoscopes that receive their illumination through cords have already been relegated to the storehouse of antiquity.

Before this Society, in Washington, in 1913, Harrower presented an instrument in which the Morton-Marple ophthalmoscope was combined with the dry-cell battery and case of the commercial pocket flashlight. This instrument, however, could be used only for the direct method, and, in the discussion of it, Marple called attention to this fact. I think the application of this form of dry cell and its case to the electric ophthalmoscope marks an advance in utility and simplicity. Dr. Hare has modified the shape of the hood containing the lenses, and has readjusted the system of lenses in the hood, while using at the same time the Marple mirror aperture and dry-cell battery which goes with the Marple instrument, as made by Meyrowitz; Hare's instrument likewise has an attachment of a very strong plus lens for the minute examination of the cornea. I consider this no advantage at all, but rather a detriment, inasmuch as sometimes it slips up in front of the eye when not needed, and so obscures the vision. The lenses in his instrument enable one to examine the cornea thoroughly. It is to be hoped that this objectionable element will be finally eliminated from his instrument. The pear-shaped hood enables one to get much nearer to the patient's face in the direct method, is more graceful and economic than that in any of the other instruments.

May has recently presented an electric ophthalmoscope, made by Wappler, in which the focusing, primarily developed by Marple, is more accurate and satisfactory. The command of the illumination is simpler and easier. Moreover, in this instrument the light from the lamp is transmitted upward through a glass rod and is reflected from a prism, not from a mirror; the aperture is circular and is placed above the reflected light. This instrument seems in some

respects to have advantage over all the others, but it is badly balanced to my hands and the prism through which the light passes is exposed so that it may be easily scratched. This I consider a disadvantage. The illumination is obtained also from the same pocket torch, first suggested by Harrower, and can be used directly attached to the instrument, or from a box to which electric cords run. I believe this latter arrangement will be ultimately eliminated. I understand Koller, likewise, has made some modification of the reflecting surfaces in the electric ophthalmoscope, but I am not accurately acquainted with it. The instrument of Marple, to my hands, likewise, is clumsy, the distance between the battery and ratchet is too great for comfort, and the whole thing has to be carried in a large box. The instrument of May, likewise, comes in a still larger box. It seems to me that this is a decided disadvantage to any instrument. Such instruments are certainly not easily portable.

The original instrument of Dennett had a circular opening, but, as stated in his letter to me, his subsequent one had an opening whose vertical diameter was greater than the horizontal, for obvious reasons. Marple changed the circular aperture to the vertical one because it gave him a better view. I, myself, have experimented with a number of apertures, and in a conversation on this subject with Dr. Dennett he suggested that a triangular segment of 60° cut in the Marple mirror would give better sight than the slit. Thinking this was too much, I divided the segment by half, making it one of 30°, and this aperture I use in the instrument I shall show you. The Marple aperture to me is unsatisfactory, and I frankly think the circular one if large enough is the best of all.

In all of our deliberations we should not forget the mechanicians who carry out our ideas. Among these Meyrowitz, DeZeng, and Wappler are to be mentioned. DeZeng has an instrument which goes by his name; it has a circular mirror and a circular aperture; it is satisfactory, except for

its clumsiness. DeZeng has suggested and used a polished metal mirror with the Marple shape and aperture, but I repeat I think the mechanical ingenuity of Wappler in developing May's instrument supersedes them all. Electric retinoscopes, likewise, are on the market, and some claim to use them with satisfaction. To me they are not satisfactory. The electric light, as well known, throws a sharp shadow, and the penumbra, upon which our judgment of the shadow in retinoscopy to some extent depends, is lacking. I believe this instrument will pass, or will certainly not be universally used.

Recently Bausch and Lomb have printed a detailed description of a large Gulestrand ophthalmoscope, binocular in character, with electric illumination, for the examination of the fundus of the eye with stereoscopic effect. It magnifies from five to forty times for monocular observation and twenty times for binocular; it is said to be free from disturbing reflections. I have not used this instrument, but doubt its practicability. If it has stereoscopic value, it will mark an advance in ophthalmoscopic work, but it can never be universal on account of its cumbersomeness. It will probably be used in hospitals and for physiologic study.

New ideas are rare, and those which surgeons present to instrument-makers are always more or less hazy and ill-defined. It is his mechanical ingenuity which renders our efforts fruitful, and to him due credit should be given.

It was my purpose to put together an instrument, composed of the parts of those that have gone before, which could be carried in the pocket, was well balanced, easily handled, and simple and reasonable in price. I therefore adopted the hood of Hare and the suggestion of Harrower, attaching the Harrower illumination to the Hare hood, and put a button upon the side of the stem, so that the focus of illumination could be altered, as the case of preceding instruments, and the instrument used in the indirect and direct method. But I adjusted the distance between the case con-

taining the battery and the hood so that the instrument was easily balanced and comfortable in the hand, and when in use the ball of the forefinger would fall upon the adjusting button. The instrument is so slender it can be slipped into a case no larger than that used for spectacles if need be. To me it is most comfortable carried in the upper waistcoat pocket.

I use the Marple mirror, but have cut out of it, as stated, a segment or triangle, the base of which is equivalent to an arc of 30°. The illumination from this to me is better than that furnished by the Marple aperture, and is completely satisfactory. I have used this instrument for four or five months daily in my office and in practice outside.

Fig. 1.

The electric ophthalmoscope has come to stay. I believe it will doubtless soon supersede the old instrument entirely, not only in hospital work but in private practice also.

I conceive that there are several epochs in the development of the ophthalmoscope, and they are marked as follows:

(1) The discovery of the principle of the reflection of light from the background of the eye. (2) The ophthalmoscope of Helmholtz. (3) The instrument of Liebreich. (4) The application of convex and concave lenses in a revolving disc behind the central aperture of the mirror. (5) The manner of adjusting these lenses, as suggested by Loring. (6) The invention of the electric ophthalmoscope by Dennett. (7) The substitution of the dry-cell battery as a means of illumination instead of the electric wire current.

To whom priority in the use of the dry-cell battery as the means of illumination in the electric ophthalmoscope is due I am not certain, but I hope this will be established by the discussion which may follow. Finally, I think it regrettable

that the name of the originator of this instrument has not been connected with its various modifications, and I suggest for the sake of justice that all electric ophthalmoscopes henceforth be designated by hyphenated names in which that of Dennett shall take precedence.

DISCUSSION.

Dr. W. B. Marple, New York: There can be no possible question but that to Dr. Dennett belongs the honor of having given us the first practical form of the electric ophthalmoscope, and that all subsequent models have been modifications of his original instrument. In 1906, when I first read a short paper presenting a model of such an instrument to this Society, I said: "In presenting this model of an electric ophthalmoscope I want to say that it is Dr. Dennett's ophthalmoscope, with two or three modifications, but these modifications, at least in my hands, make it a much more useful instrument."

Dr. P. A. Callan, New York: I had an electric ophthalmoscope, but the metal mirror has undergone oxidation. When I look through the upper part at the light, I get atrophy of the optic nerve. It is bluish, in other words, and cuts off the red rays. The gentlemen should be on their guard against looking through an oxidized mirror or they will make a faulty diagnosis.

Dr. S. Lewis Ziegler, Philadelphia: In reply to Dr. Callan's criticism of the metal mirror, I would like to say that it is very easy to clean an oxidized mirror. If that is the only objection he has to the instrument, however, it is readily removed, since I am now using a new type of metal mirror that is absolutely non-oxidizable. I am sure that the non-breakability and avoidance of annoying reflexes are two great advantages. The metal mirror is U-shaped, and follows the same form as Dr. Marple's. The sliding adjustment of the lens should be credited to Dr. Dennett, as Dr. Claiborne has stated, because in his original paper before this Society (1885) Dr. Dennett describes and illustrates this attachment, which was made for him by Meyrowitz. The instrument that I presented before this Society last year

was chiefly a modification of the working parts for the purpose of securing a more practical and efficient instrument, of lighter weight, and freed from reflexes. The ophthalmoscope itself possessed a novel arrangement of lenses.

In answer to Dr. Claiborne's query, I would state that Dr. Crampton, of Philadelphia, was the inventor of the battery in the handle, and should, therefore, have the credit of this useful improvement.

DR. J. HERBERT CLAIBORNE, New York: I wish to call attention particularly to the shape of the hood in Hare's instrument. The shape of the hood in the instrument containing Loring's quadrant and system of lenses is round, and has a semicircular projection forward which is practically useless and serves only the purpose of exploiting the name of the maker or inventor. Hare's hood is pear-shaped, as shown, is slender and graceful, and one is able to approach nearer to the patient's face in using it. These two characteristics I think mar the usefulness of the May-Wappler instrument likewise.

I think it would be well if all instrument-makers were to discard the circular hood, with the unnecessary prolongation, and adopt instead the Hare hood, with its system of lenses. This would make for grace, economy, and effectiveness.

A SIMPLE MODIFICATION OF THE BERGER LOUPE.

DR. EDWARD A. SHUMWAY, of Philadelphia, exhibited a simple modification of the Berger loupe, which consists in the attachment to its side of a brass clip to hold the small electric lamp that has been placed on the market recently, which has the appearance and size of a stylographic pen. The lamp has a small, one-cell battery, which illuminates a Tungsten filament lamp, and has a permanent turn-on arrangement. It can be purchased at any of the electric supply houses or instrument shops. The light-rays from the lamp are projected toward the focal point of the Berger loupe, and give sufficient light to satisfactorily illuminate

the eye under examination. It has been of especial value in the removal of foreign bodies from the cornea, and in pulling out delicate misplaced cilia from the eyelids, as it gives an independent source of illumination, and leaves

Fig. 1.—Simple modification of the Berger loupe.

both hands free for manipulation of the eyespud and for holding the patient's eyelids. It has also the advantage of being independent of wires to batteries or rheostats, and can be readily removed from the loupe when illumination is not required. The Zeiss loupe has also a lamp to illumi-

nate the eye, but the instrument is much more expensive, the lamp requires a separate battery or rheostat, and moreover the focus is rather short (1¼ inches), while the Berger loupe focuses at about two inches, giving more space in which to move the spud or other instrument. Other arrangements have been employed to illuminate the eye in removing foreign bodies, but they are not sufficient when the operator requires binocular magnifying power to assist his near sight, and self-illuminated spuds have been unsatisfactory, in my experience, as the illumination is not constant but varies with the distance of the spud from the eye. Moreover, it is very difficult to get patients to hold their eyes still, as they seem to fear a shock of some sort when the light from such an instrument, connected by wires to a battery or rheostat, approaches the eye. The ordinary Tungsten lamp furnished with the stylographic pen battery has a plain glass top. For more powerful illumination I have replaced it by an imported lamp, which has a small magnifying lens fused on the top, by means of which the light-rays are better concentrated. This makes the apparatus more useful in dispensary work, especially in the examination of inflammatory conditions of the anterior portion of the eyeball or its adnexa, making it usually unnecessary to take patients to a dark room. The loupe has been cut out on each side to remove the obstruction to lateral vision, of which complaint is occasionally made, but this is not a new feature. The modified apparatus can be obtained from Wall and Ochs, of Philadelphia.

DISCUSSION.

Dr. W. B. Marple, New York: Two or three years ago, at the Oxford Congress, I referred to the use of the electric ophthalmoscope in removing foreign bodies from the cornea. I have had the Berger loupe in my office for many years, and formerly used it a great deal, but I rarely do so now because, with the electric ophthalmoscope and with a convex lens of 12 to 20 D. back of the aperture, the minutest

foreign body can be promptly differentiated as a black speck on the red reflex from the fundus. While the eye is thus illuminated with the ophthalmoscope in one hand, and the foreign body is kept in view, with the other hand the spud is held and the foreign body readily removed. The observer can tell at once when the offending particle is gone. Since employing this method I have never used the Berger loupe.

DR. E. A. SHUMWAY, Philadelphia: This apparatus has the advantage of leaving both hands free. No assistant is then necessary. The lids can be held with one hand, while the spud is manipulated with the other, and the lighting apparatus is entirely independent of wires, so that the surgeon may walk freely about.

NAGEL'S ANOMALOSCOPE FOR TESTING COLOR-VISION.

CHARLES H. WILLIAMS, M.D.,
Boston, Mass.

Just before the war I was able to get from Franz Schmidt and Haensch, of Berlin, one of Nagel's anomaloscopes for testing the color vision, and the use of this instrument during the past year has shown that it is a valuable addition to our methods for testing the color sense, especially in the determination of doubtful cases. On account of its cost and delicacy the instrument is not well adapted to the routine examination of large numbers of men at many different places, but it should be available at some central station where the reëxamination of doubtful or appealed cases can be made.

In this instrument only pure spectrum colors are used, and a record of the examination can be made in definite form which can be referred to or reproduced at any time.

The principle of the instrument depends on the fact shown by Rayleigh that, when a normal eye looks at a colored field

of which one half is uniformly lighted by a pure yellow light from about the sodium line, and the other half by a mixture of red from about the lithium line and a green from about the thallium line, there will be found a certain mixture of the

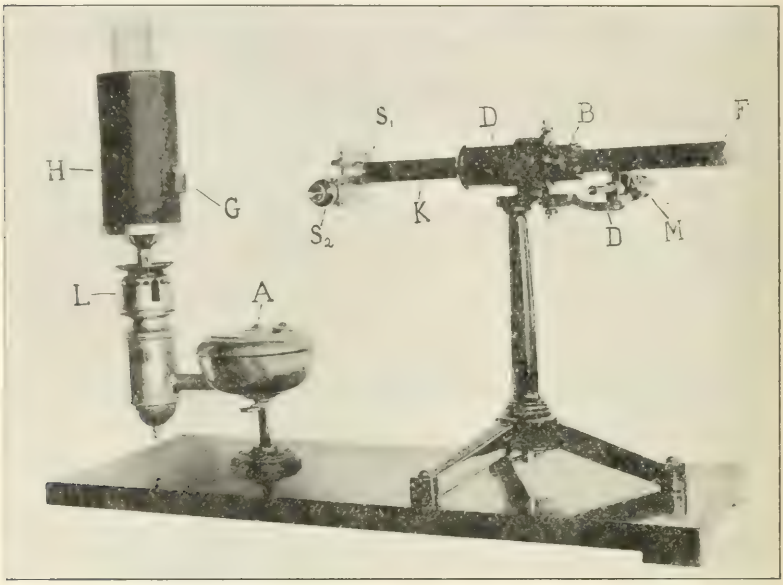


Fig. 1.—Nagel's anomaloscope for testing color vision.

K, Collimator tube; F, eye-piece tube; D, prism; M and lower D, screws to control position of eye-piece tube; B, diaphragm, to alter size of visual field; A, holder for alcohol; L, mantle lamp using alcohol vapor; H, asbestos chimney surrounding glass chimney of lamp; G, ground-glass plate, source of illumination for anomaloscope; S_1, right-hand screw (G_1 of Fig. 2), controlling width of upper slit and lower half of field as seen at the eye-piece F. This screw regulates the brightness of the pure yellow half of the field. S_2, left-hand screw (G_2 of Fig. 2), controlling the width of the two coupled slits and the upper half of the field as seen at the eye-piece. Through one slit light is transmitted through the prism D, which, when seen at the eye-piece, corresponds to the lithium red; through the other slit comes the thallium green. By moving the screw, S_2, the upper half of the field is illuminated by a proportional amount of the red-green mixture, from a red to a yellow and then to a green.

red and green which gives a yellow that cannot be distinguished from the yellow of the sodium light when the intensity of the sodium light is adjusted to a certain brightness.

Such a normal eye cannot make a match between the pure yellow and the mixture of red and green by any change in the brightness of the yellow whenever the proportion of red or green in the mixture is so great as to give the mixture a red or green color, but the eye which is defective in its color sense will be able to make such a match by changing the brightness of the yellow. The arrangement of the anomaloscope is seen

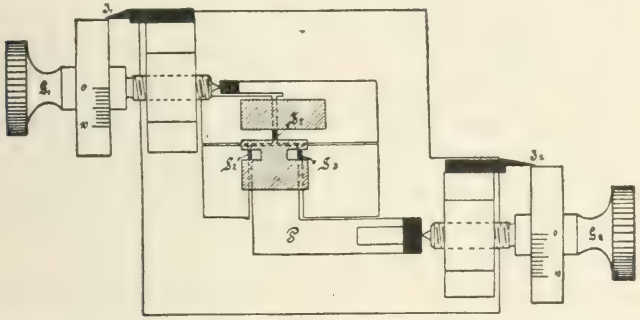


Fig. 2.—Slits on front end of Nagel's anomaloscope when looked at from the position of the lamp. (From Köllner, Die Störungen des Farbensinnes, p. 70.)

G_1 (shown at the upper left-hand side) is the right-hand screw (shown in Fig. 1 as S_1), which governs the width of the upper slit S_1 and thus changes the brightness of the yellow in the lower half of the field. G_2 is the left-hand screw (shown in Fig. 1 as S_2), which governs the width of the two coupled slits S_2 and S_3 through which the red and the green light respectively comes to make the mixture of red and green as seen in the eye-piece of the anomaloscope in the upper half of the field. These two slits are coupled together so that one can be changed only with a corresponding change in the other. The total amount of the width of both slits, the space between them, and their position relative to the prism of the instrument, remain unchanged. By means of this second screw S_2 any desired mixture of red and green can be obtained, from a pure red, through the yellow, to a pure green on the other end.

in Fig. 1. When the screw S_2 is turned to the limit in one direction, only a pure red is seen in the upper part of the field, and by turning it in the opposite direction to the limit only a pure green is seen, and between these positions any proportional mixture of red and green can be made.

The arrangement of the slits and controlling screws can be better understood from Fig. 2.

In his paper* Nagel says: "The color blind get the matching with yellow (by altering the intensity of the yellow) by every sort of proportional mixture of red and green, and even with a pure red or green, and are thus sharply differentiated from every kind of trichromats. The red-blind (protanopes) can be easily separated from the green-blind (deuteranopes) because they must give a different brightness to the yellow in order to obtain a match."

A defective eye will sometimes match a mixture of the red and green, which to a normal eye appears yellow, with a yellow of a certain brightness, but the normal eye will not be able to get any match between the two halves of the field by varying the intensity of the pure yellow in the lower half when the upper half shows a decided preponderance of red or green in the mixture, whereas the defective eye will easily find such a match.

As stated by Köllner,† it will be found best to make the tests in the following order:

1. In the upper part of the field a pure red; see if it is possible to get any match between the two halves of the field by changing the brightness of the yellow.

2. Place the screw S_1 so that the sodium yellow, lower half, corresponds to the brightness which the normal eye matches with the red-green mixture (when set at about 62, yellow, for screw S_2) in the upper half of the field (usually at about 14 on the scale of screw S_1), and see if a match can be made between the two halves by turning the screw S_2 so as to vary the red-green mixture. This can be done in most cases, but not in all.

3. Place the left screw S_2 so that it is about 15 numbers lower on its scale than the normal (about the average number for the deuteranope), and see if a match can be made by varying the intensity of the yellow with the right screw S_1.

* *Zeitschr. f. Augenheilk.*, xvii, No. 3.

† Die Störungen des Farbensinnes, Dr. Hans Köllner, p. 72.

4. Place the left screw at about 10 numbers higher than the normal (about the average for the protanope), and see if a match can be made by changing the intensity of the yellow with the right screw S_1.

THE MAY ELECTRIC OPHTHALMOSCOPE.

CHARLES H. MAY, M.D.,
New York City.

The essential advantage of the ophthalmoscope, and the one in which it differs and is superior to all other luminous instruments of this sort, is the substitution of a solid rod of glass acting as a condenser at its lower part and as a reflecting prism at its upper part, with the addition of other converging surfaces sufficient to gather the rays with which the fundus is lighted up, so that there results a solid illuminated surface. This is the main improvement and it is a marked one.

In the ordinary reflecting ophthalmoscope a stationary round or a tilting oblong concave mirror is used to reflect the light from the source of illumination into the eye; good results ensue only when the source of illumination is solid; hence we use an Argand burner, or if we employ an electric lamp we must get rid of the film effect and use a spiral film with frosted globe; we all know how unsatisfactory the ophthalmoscopic examination is when an electric lamp is used with the ordinary film. An objection to all electric ophthalmoscopes which are founded upon the principle of using a mirror, whether glass or metal, to reflect the electric light contained in the stem of the instrument, such as was introduced by Dennett, is that the illuminated area of the fundus will always have the form of the lamp filament. In my device this is obviated and an even, solid collection of rays is thrown upon the background, free from shadow, reflex, or suggestion of lamp filament.

The other advantages claimed for my electric ophthalmoscope are merely matters of convenience. The instrument is small, unbreakable, goes into a case which, including the battery handle, can easily be carried in the pocket. There is no mirror requiring care against breakage. With a small connecting piece known as an adapter the ophthalmoscope can be operated by *any* flashlight, whether of the flat design, fountain-pen shape, or any other form or size; it must be remembered, however, that the smaller the size of the dry cells used, the shorter the life. The instrument can, when desired, easily be converted into an ordinary reflecting ophthalmoscope with tilting mirror.

The instrument is provided with a rheostat on its stem; this is of value in protecting the small 0.1 ampere lamp against excessive current, especially when used with wall outlet with controller. It can be used with either a battery handle, any two-cell flashlight, or with cords. A special battery handle provided with its own rheostat will be found very convenient. I find it satisfactory also to use a rheostat on the handle when cords are used.

The lens-disc portion of the ophthalmoscope is of the double-disc variety introduced by me in 1900, in which a large number of lenses can be employed, rotated over the sight-hole without the interposition of a troublesome supplementary quadrant; one disc (the anterior) contains the convex lenses, and the other (the posterior) the concave lenses. This instrument has other optical advantages, such as thinness and the existence of a sharp beveled edge overlapping every other aperture, thus reducing to a minimum the possibility of reflexes from the openings in the plates. Since the general practitioner does not require so many lenses, the instrument is also manufactured with a single lens-disc, with corresponding reduction in cost.

By freeing the lamp, removing its metal cap, and covering with a small cylinder of rubber tubing, an excellent transil-

luminator is provided—one with which the pupil can be illuminated when the end of the instrument is applied to the external surface of the lids; it is not necessary to apply the lamp directly to the sclera.

In conclusion, I wish to repeat my claim that this system of illuminating the background is superior to the one now in use, in which a reflecting mirror is employed, and that it lights up the fundus much better, and is free from shadows, reflex, or lamp-film effect.

I have presented this electric ophthalmoscope on previous occasions, but not to this Society; and since its original presentation it has been modified and some of the accessories improved.

DISCUSSION.

DR. S. LEWIS ZIEGLER, Philadelphia: I should like to ask whether the principle of Dr. May's instrument bears any resemblance to the prism-rod of Dr. Black, of Milwaukee.

DR. F. H. VERHOEFF, Boston, Mass.: In some respects I think it is an advantage to have a picture of the filament when using an electric ophthalmoscope. Haab long ago pointed out the value of indirect illumination in the case of an ordinary ophthalmoscope, and I find it of equal value in the case of the electric ophthalmoscope. You throw the image of the light source a little to one side of the area you wish to examine, and you can then make out details that you cannot see when the whole fundus of the eye is evenly illuminated.

DR. C. H. MAY, New York: I regret that I cannot answer Dr. Ziegler's question, since I have not seen nor read of Dr. Black's prism-rod. In replying to Dr. Verhoeff, I would say that there is undoubtedly an advantage, at times, in exploring minute details with subdued illumination; this is accomplished with my instrument by using the rheostat and reducing the intensity of the illumination to the requisite degree without removing the ophthalmoscope.

STRONGER EYE MAGNETS.

WALTER B. LANCASTER, M.D.,
Boston, Mass.

My experimental work on eye magnets was done about fifteen years ago. I learned a number of things about magnets, but I found that none of them was new. All the facts had been discovered by previous workers. If I made eye magnets that were stronger than those before in use, it was not because I discovered any new facts, but simply because I applied in a different way principles that had long been established. Two papers were read before the New England Ophthalmological Society, but not published.

Years have passed, and much has been written on eye magnets since that time, but still no one seems to have made the same application that I did of these old-established principles. Last year I showed a magnet tester at the meeting of the American Medical Association. Tests with this, of eye magnets at several large eye clinics in different cities, have shown that the average eye magnet in use is less than half as powerful as it might be.

Without some such instrument it is not easy to compare the power of different magnets, because hardly ever have two makers of eye magnets tested them in the same way or in ways that were comparable. It is obvious that the test-object should be small, and that the tests should be made at such distances as the magnet is intended to be used. For example, a small hand magnet, intended to be used at short range to remove fragments usually weighing much less than 0.1 gm., should not be tested at a distance of 10 or 20 mm. or with a large test-object. Again, the test-object should

be preferably spheric, as the shape has a very important effect on the strength of pull, and no shape is so easy to duplicate and so fair as the sphere. I shall be glad to send the testers for a trial to any one who wishes to test his eye magnets. Perhaps there are others stronger than mine, but I believe I am not exaggerating when I say the *average* eye magnet is not half as strong as it might be. Hirschberg says: "Any one who undertakes to do magnet operations should be properly equipped—one does not do cataract extractions with a pocket knife." This was apropos of a magnet sent to Hirschberg from a large eye clinic with the request that, if found deficient, a new one be sent. Hirschberg says it compared to his own like a cross-bow to a breech-loader.

The original purpose for which my testers were made was not to compare different magnets, but to have at hand a ready means of trying our magnets from time to time to see whether they were maintaining their efficiency. The importance of doing this is obvious.

Let us consider what determines the strength of eye magnets in order that we may discuss intelligently some of the errors in design which have made eye magnets needlessly inefficient. It is not necessary to go fully into the physics of the problem. Any one who is interested can find much more complete details than would be possible in a paper like this by consulting recent text-books on physics or the monographs in the "Encyclopædia Brittanica" or "The Electromagnet" by S. P. Thompson—the last is very complete and is easy reading.

We need to be familiar with the conception of lines of force introduced by Faraday. A compass needle placed near enough to a magnet to be influenced by its action, *i. e.*, anywhere in its field, will turn and place itself parallel to the lines of force at that place. Imagine a great number of very small needles each free to move on its axis: these would

arrange themselves along the lines of force. Iron filings do this and form the well-known picture. To speak more exactly, each line of force represents the path which an isolated pole would follow if free to move in the field of a magnet. A north pole would move one way along the line of force, a south pole would move with equal force in the opposite direction along the same line. In representing these, one line is drawn or imagined for each unit of magnetic force, so that where magnetism is intense, the lines are more numerous and near together, and where it is weak, the lines are farther apart. Thus given the lines of force, and we know at once the strength and direction of the magnetic force.

This tendency of a needle free to move to place itself parallel to the lines of force is shared by the molecules of a magnetic substance. Each molecule is supposed to have a permanent polarity. When a bar of iron is magnetized, the molecules tend to arrange themselves with north poles pointing the same way; the more strongly the iron is magnetized, the larger the proportion that are thus arranged; if all were thus arranged, the bar would be saturated. In the case of steel, once they are made to assume the position of parallelism, many of the molecules stay in that position, thus forming a permanent magnet. Repeated jarring tends to destroy the permanency by encouraging the molecules to return to their original position. In the case of soft iron, the molecules are freer to move, and they return at once to their original position without jarring when the magnetizing force is removed.

A second conception needed is that of the magnetic flux or circuit. All are familiar with the idea of electricity flowing through a wire or other conductor. Similarly, there is a flow of magnetism around a circuit, out the north pole, around through the air or other material, and into the south pole of the magnet, then along its core to the north pole, and

so on. The lines of force represent the path of this force. It is also called magnetic induction. As there is a resistance to the flow of the electric current, so there is to the flow of the magnetic current, but to avoid confusion it is called *reluctance.* Similarly, the magnetomotive force of a magnetic circuit corresponds to the electromotive force of an electric circuit. The amount of magnetism then depends on the magnetomotive force and on the reluctance of the circuit. These are the two things we wish to know about in order to understand what determines the strength of an eye magnet.

Of these, the magnetomotive force depends on just two things—on the number of turns of wire in the coil around the magnet, and on the number of amperes of electricity flowing through this coil; briefly, on the *ampere turns.* The only limit to the magnetizing power of a coil is the number of ampere turns. This is limited by the size we can permit, and for a given size there is a limit to the number of amperes we can use, depending, first, on the limitations of our electric supply and the wiring of the building, and, second, on the heating of the coil. The quantity of heat generated per unit of time in a coil of given dimensions increases as the square of the number of amperes of current—double the number of amperes and you get four times the heating effect if the wire remains of the same size. The magnetizing force then depends solely on the ampere turns, and for a given size of coil the number of amperes allowable depends on the heating.

So much for the magnetizing force. But the amount of magnetism depends not only on the magnetizing force, but also on the reluctance in the magnetic circuit. The reluctance of air is taken as unity. All other substances and vacuum have *about* the same reluctance except iron and a few other metals and alloys. Iron has very much less reluctance than air. For weak magnetization several thousand times as many lines of force (units of magnetism)

would pass through iron as through an air path of the same dimensions. But as the magnetization increases, the reluctance of iron also increases (different from electric resistance) until, with very high magnetization, it offers about the same reluctance as air. This is the point of saturation. As with electric conductors, the larger the cross-section of a magnetic path, the less the reluctance, and the longer the path, the greater the reluctance.

All this is very simple and elementary. It is when we come to apply it that errors seem to be made. It will be worth while, perhaps, to take up some of the more prevalent and misleading errors.

The intensity of a magnetizing force is represented by the number of lines of force, but this does not determine the strength of pull of a magnet on a piece of iron in its field. One may have a very intense magnetization, with little or no tractive power. If the field is uniform, there is no tendency for a fragment to move except to turn and place its long axis parallel to the lines of force. A fragment tends to move from one part of a field to another stronger part, and to move in the shortest path to that stronger part of the field. If the field is of uniform strength, there is no stronger part for it to move to, and so it remains where it is. We can understand this if we remember that a fragment always has two poles, north and south. One is attracted and the other is repelled. If the field is uniform, each is acted on by a force of the same intensity, and therefore a couple is formed which turns the fragment on its axis until it is parallel to the lines of force, but has no tendency to move it from its place. This is why a compass needle floating on a piece of cork has no perceptible tendency to move as a whole toward the north pole or the south pole of the earth, because in the small space of a room the lines of force of the earth's magnetism are sensibly parallel—field is uniform.

To secure a strong tractive power, then, we want a magnet the field of which is very intense at its pole, but with the lines diverging strongly from that point, so that the field is weaker —much weaker—beyond. One pole of the fragment we wish to pull will then be in a very much stronger part of the field than the other, and so the former is pulled much more forcibly than the latter is repelled. In defending the Innenpole, or ring magnet, Mellinger has laid great stress on the fact that the lines of force in his field in the ring are parallel, and not widely diverging and scattered, as with the ordinary bar magnet. As a matter of fact, the lines of force in the ring magnet are parallel for a short distance at the midplane of the ring *when there is no iron there*, but as soon as you introduce the iron style which is the pulling instrument, this parallelism is changed. The lines of force converge toward one end of the iron rod or style and diverge from the other end. If they did not, the style would not have any attractive force. The reason the lines of force flock toward the iron is because it offers so much less reluctance to the magnetic flux. It also increases the flux for the same reason. The larger it is, the more it increases the total flux. If the whole of the ring were filled with iron instead of being nearly all air, the increase would be very great. A ring or solenoid with a bar of iron filling it is a much stronger magnet than one without the iron or with a small piece only partly filling it.

What we really should aim to devise is a magnet with a field in which the lines of force have the distribution which will produce in the highest degree the effect we desire. We see at once that it is impossible to have the same magnet and pole piece produce the highest efficiency in both action at a distance and pull in contact. The two properties represent two distinct problems of design. Without going into the geometric principles involved, it may be stated that the pole for a magnet to act at a distance should not be slender and sharp pointed, but blunt and obtuse, so as to project the

lines of force to a greater distance before they widely diverge. This is not purely theoretic. I made many measurements on the effect of different-shaped pole pieces. Starting with a long, tapering cone, I made a set of magnetic measurements and then turned the pole down in a lathe to a shorter cone and repeated the measurements, and so on. I also used, besides the cone, pole pieces of hyperboloid and bullet or Gothic-arch shape of different proportions. The blunter and shorter the pole, the stronger the pull when tested at 20 mm. or more. In the figure (Fig. 1) it is easy to see that the shape represented by the full line contains more iron, more carrying power, and more forward projecting power than the shape represented by the dotted line. Or, look at it this way: suppose the space between the dotted line and the full line filled with air in the one case, with iron in the other. Of course, the iron has far less reluctance than the air. Therefore it will carry more lines of force on toward the tip than when its place is taken by air.

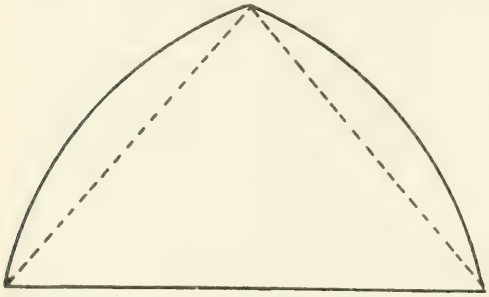


Fig. 1.—Shape of pole suitable to give strong pull on fragment at a distance.

Another prevalent error concerns the direction of pull of a magnet. It is a common and natural mistake to suppose that the direction of pull is along the lines of force, whereas it is not usually so, but is oftener at an angle to them and may be square across them. The law is that the path followed by a fragment free to move in a magnetic field is toward a more intensely magnetized part of the field by the shortest possible path. This was pointed out by Sir William Thompson (Lord Kelvin) in 1847.

Core.—The usual material for the core is soft Norway iron. Much work has been done in the study of different materials because of the importance of the subject in dynamo construction. A very pure steel has been found slightly superior even to soft iron when high degrees of magnetization are used ("Encyclopædia Brittanica," eleventh edition, vol. xvii, p. 228). I do not know of it being used for eye magnets.

The Haab magnet illustrates a common fault of cores. It is too short for its thickness. It has long been known that short, thick cores did not have as powerful a pull on objects at a distance as longer cores. There are certain advantages in a short, thick core in making some electric machines, but not for eye magnets. A good proportion is 10 or 12 times as long as it is thick.

In his latest model Haab adheres to the short, thick core, but he adds a large expansion of the posterior pole in the form of a bell-shaped iron cover for the coil. It was long ago shown that a magnet with the posterior pole expanded in any way with iron would exert a stronger pull. Therefore, Bradford made use of this device, as did one of the English designers before him; so did Volkmann, more recently, by having the posterior end of his core extend far beyond the coil. I have seen measurements reported where the pull was increased sixteen fold by this device (Thompson, "Electromagnet," pp. 140–149). I tried it years ago, when experimenting on this matter, but when I measured the pull with and without such polar extensions, I found no gain whatever. This puzzled me a good deal at first, but on further experiment I found that, if I used a very weak current and therefore a weak magnetization, the polar extension made a considerable difference. With strong currents it did not. But to-day no one designs an eye magnet to be used with a weak current; therefore this device is worthless, and I abandoned it. (For explanation, see Thompson.)

Another mistake is the attempt to increase the power by

making the core tapering—larger at one end than the other—as Volkmann and others have done. The greatest amount of magnetic flux for a given weight of core will be obtained when the cylindric shape is used. I made no experiments on this myself, because I found reports quoted in Thompson's book which are sufficiently convincing.

Coil.—Since the magnetizing force depends on the number of turns in the coil multiplied by the number of amperes, it was a natural assumption that, by making the coil consist of more layers and therefore more turns, greater power would result. About every writer on eye magnets has fallen into this trap. If half the layers were removed from each of the two coils of the Haab and similar magnets, the result would be an actually stronger magnet. The reason is simple enough. The outer layers of the coil are much longer than the inner, so that if half the layers were omitted, much more than half the wire would be removed, and the remaining portion would have much less than half the resistance of the original large coil; hence, much more than twice the number of amperes would flow through it. For instance, suppose there were 28 layers of 100 turns each, the inner 14 inches, the outer 36 inches long, the average 25 inches long. There would be 2800 turns and 70,000 inches of wire. Suppose the resistance equaled 1 ohm per 3500 inches of wire, then the total resistance would be 20 ohms, and on the usual 110-volt circuit we should get 5½ amperes of current: 5½ A. × 2800 T. = 15,400 ampere turns. With 14 layers, inner 14 inches, outer 25 inches, average 19.5 inches, 1400 turns would be 27,300 inches long, and resistance would be 7.8 ohms; this would give, on 110-volt circuit, 14.1 amperes. Multiply by 1400 to get the ampere turns, and we get 19,740 instead of 15,400, a gain of between 25 and 30 per cent. If the process were repeated and half the remaining coil removed, the strength would be again increased. The rule, then, for giant magnets is: use as few layers in the coil as your electric

supply and the heating effect will permit. The rule for size of wire was pointed out by Clerk Maxwell many years ago: "The magnetizing power of a coil, supplied at a given number of volts pressure, is independent of its length, and depends only on its gage." That is, use as large a wire as you can, within the limitations of your electric supply and the heating effect. For example, in designing the large magnet for the Massachusetts Charitable Eye and Ear Infirmary, I was limited to 30 or 40 amperes of current at 220 volts. I used a No. 8 double cotton-covered wire and wound on enough layers to give 6 or 7 ohms resistance. If I could have used No. 7 with the same or fewer layers, I should have secured greater magnetizing power, but it would have meant 50 or 60 amperes, because the resistance of the larger wire would be less.

Heating.—The first thing an electrical engineer thinks of in designing a machine with a coil is the heating factor. He has well-known and tried formulæ for this, and when consulted about an eye magnet, he applies these formulæ. Haab, Hirschberg, Volkmann, and many others state that their magnets were designed by electrical engineers or physicists or that they were consulted. I consulted several. All assured me, with various degrees of positiveness, that my plans were not feasible. The reason is plain. They were thinking of the conditions under which their magnetic machines are operated, not of the conditions under which an eye magnet is used.

The main consideration is the time element; it takes time to heat up. If we wish to use a strong current without overheating, we must allow it only a short time to act. Fortunately, it requires only a short time for a magnet to pull out a foreign body. To be sure it takes time in many cases to dislodge and start the foreign body. Often a minute is enough, seldom over two or three minutes' *actual pulling.* Of course, more time is required to manœuver to get patient and magnet tip just where you want them, but

after that is done and the current turned on, more will be accomplished in one minute by a strong magnet than in ten minutes by a weaker one.

I therefore designed my magnets—both the small and the large—to carry such large currents that, inside of ten minutes, they would become dangerously overheated. There is no need of keeping a steady pull on a fragment in an eye for more than a minute or two, though this often has to be repeated several times from various angles. Of course, there is no advantage whatever in keeping the current on when getting ready or between tries—quite the reverse. I do not for a moment deny that there are objections to overheating, but I deliberately choose the overheating in order to get a very strong magnet in preference to not heating with a much weaker magnet. We have tried it fifteen years.

Another easily understood error is the winding of the wire in two large coils at the ends of the core, with a space between where there is no coil, as in the Meyrowitz and old Haab, but not the new Haab, models. It was long ago shown that the best way to wind a core for maximum pull is along the entire length of the core. There is less leakage and you get many more turns of wire, because the same length of wire can be wound more times around a core if distributed over the entire length than if heaped up at the ends. No doubt the reason the first Haab was wound in this way was to facilitate the mounting, but it is a serious error.

One more detail is of sufficient importance to be pointed out. It is essential, if we would get the maximum pull, to be sure that the winding of the coil extends to the very end of the core. This is one of the details that the manufacturer is likely to change for his own convenience. Haab speaks of the importance of this, but yet his latest model errs notably in that direction. It will be found that all thick, bulky magnets err in this particular, because the bulky coil is in

the way, and to get free space enough to work satisfactorily on an eye the core and pole piece *must* project more than would be necessary with a more slender, less bulky instrument.

Leakage.—It is in the care exercised to avoid every little source of leakage that the fine points of magnet design and construction are shown. By leakage is meant the loss by magnetic flux (lines of force) escaping before the end of the pole is reached, and passing around through the air to the opposite pole, without contributing to the effective power of the magnet. Or, we may say, that all lines of force that do not contribute to the intensity of the magnetic field where it is wanted, either by going there themselves or forcing other lines to go there by crowding them in that direction, are wasted. They have leaked away because they have found an easier path to follow. Invariably the lines of force—the magnetic flux—follow the path of least reluctance. Some of the common forms of leakage are: Too long a space from coil to working end of the pole—lines of force escape in enormous numbers from all parts of the core beyond the coil, so it is of great importance to make this as short as possible. Corners and edges favor leakage, so there should be no corners, but the core should pass smoothly into the pole without a break. Making the hole for the pole piece to screw into in the end of the core deeper than the length of the screw that is to fill it leaves a void filled with air, and therefore of greater reluctance, and so forces lines of force out of the core which otherwise would have found their easiest path to lie farther forward into the pole, where they would have been of use. As there is always some void at this point, it is well to make the screw of small diameter, thus reducing the size of this space. It is important that the pole piece taper continuously, but not necessarily uniformly. A constriction followed by a bulging is very bad indeed. There are some advantages in a flattened tip which

can be introduced through a linear cut, and when turned will hold the incision open, but, on the whole, the most useful shape is circular on cross-section. A bent or angular tip is a great advantage in many cases, in spite of the fact that one loses a little in pulling power. The two most useful tips are shown in Fig. 2. On giant magnets there is no need for any but the shape shown in Fig. 1, because one never introduces the tip of a giant magnet into the anterior chamber. Its field of usefulness is for action at a distance. The very end of a tip intended for use in contact should be rather sharp. The cup-shaped end devised by Parker to prevent the fragment from being brushed off by the lips of the incision sacrifices something in power. Dr. Crampton has suggested that the advantages of the cup shape might be preserved with the pointed tip by encircling the tip with a sleeve of non-magnetic metal.

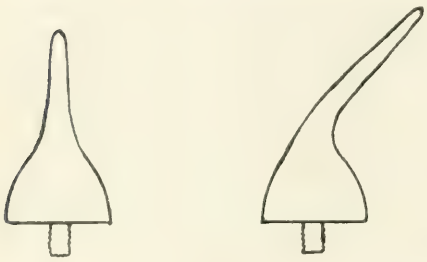


Fig. 2.—The most useful shapes for small magnets intended to act at close range; fragment in contact or nearly so.

Lately copper wire, insulated by a coating of enamel instead of cotton or silk, has become available. It has the great advantage of being fire-proof, so that it will not be spoiled if some one greatly overheats the coil. It is recommended for small magnets only, because on large wire the enamel would be likely to crack in winding.

To regulate the strength of the current a rheostat is often advised. For the giant magnet it is superfluous. It has been abandoned by Haab. The foot-switch and the regulation of strength of pull by the distance of the eye from the tip are sufficient. For a hand magnet a resistance of some sort is necessary to reduce the current. There are plenty

of good ones at moderate prices, especially those made for theater dimmers.

It is also an advantage, though by no means indispensable, to have an ammeter to show how much current is flowing through the coil. This adds $10 or $15 to the expense of the equipment.

Lastly, let me urge as strongly as possible the necessity of a foot-switch not only for the giant magnet, as urged so forcibly by Haab, but for the small magnets also. Its advantages are incontestable. One can use much stronger currents without overheating, and one has the magnet far more perfectly under control. Are there any disadvantages?

To recapitulate: The strength of pull depends not on the intensity of the magnetic field, but on the difference in intensity between neighboring portions of the field.

Direction of pull is not necessarily along the lines of force, but toward a stronger part of the field by the shortest path.

Limit of strength of pull is not the saturation point so often referred to by writers on eye magnets. The intensity of pull continues to increase as long as the magnetizing force (ampere turns) increases, though at slower and slower rate.

Cores should not be short and thick, but 10 to 15 times as long as they are thick. They should be cylindric, not tapering, and project very little beyond the coil before the tapering pole piece begins.

Coil should be wound over the whole length of the core, not with a vacant gap in the middle. It should consist of much fewer layers than is common, and be of fairly large wire, so as to carry a large current.

The risk of overheating is greatly exaggerated. It is far outweighed by the advantages of using strong currents. Overheating can be avoided by short-time exposure and safeguarded by enameled wire.

Foot-switch is indispensable.

The pole should taper in the form of a pointed arch, and be obtuse for action at a distance, sharp at the end for power in contact.

Rheostat is necessary for small but not for giant magnets.

DISCUSSION.

DR. WILLIAM M. SWEET, Philadelphia: The magnet which I use is designed upon the same general plan as indicated by Dr. Lancaster. The base of each of the points has a diameter equal to that of the core, but the shortest tip is less rounded than that shown by Dr. Lancaster. While it is true that the more nearly the tip corresponds throughout its length to the width of the core the greater the attractive power, the less rounded tip gives the surgeon an unobstructed view of the field of operation, which is a decided advantage. Last year a gentleman visited Philadelphia and tested with Dr. Lancaster's measuring instrument three magnets of the type I use, and found that my office magnet was stronger than those of similar make in the hospitals. I think this is explained by the careless use the latter have had, resulting in excessive heating and injury of the winding. I have always preferred to leave to an assistant the switching on and off of the current, so that the surgeon could give his entire attention to the operation, and therefore employ a hand-switch, although many of my magnets are supplied with a foot-switch.

DR. WALTER B. LANCASTER, Boston, Mass.: In answer to Dr. Sweet's objection to the foot-switch, I might say that in a magnet intended for the use of a number of operators it is desirable to have a foot-switch. If a man does not want to use it he need not. He can direct his assistant to open and close the circuit, as Dr. Sweet prefers to do; but of course the assistant can do this just as well with the foot-switch as with a hand-switch. If there is a foot-switch available, those who prefer to have the current under their own immediate and instant control can use it.

DR. MARPLE: What is the size of this magnet?

DR. LANCASTER: The largest magnet is one meter long and 15 centimeters in diameter.

DR. MARPLE: What does it weigh?

DR. LANCASTER: About 150 pounds. It is a little heavier than the Haab magnet.

A MORE EXACT METHOD OF MEASURING FIELDS OF VISION AND ARCS OF ROTATION.

LUCIEN HOWE, M.D.,
Buffalo, N. Y.

Several years ago I made a considerable number of observations to determine the value of the ordinary perimeter used as a tropometer to measure the arcs of rotation outward and upward, the readings being compared with other readings made with the tropometers of Nicati and Stevens. The results obtained at that time are of no special interest in this connection, but that study did show that the ordinary perimeter is a very imperfect instrument, and also that the sources of error could, with certain improvements, be largely eliminated. Since then the construction and methods have been improved, so that now it seems worth while to call attention to improvements which have the following objects:

1. To bring the center of the observed eye to the center of the arc.
2. To keep the head and therefore the eye in that same position.
3. To furnish a test light of suitable intensity and color.
4. To exclude rays from the test-object which give rise to confusing reflections.

Let us consider these in order, regarding the instrument, at first, as a perimeter only.

Probably the most frequent source of error is that the center of the observed eye cannot easily be brought just to the center of the arc. In using most perimeters the eye is simply brought near to a projecting point, which is supposed to be a little distance in front of the center of the arc. There is no arrangement, however, for locating the position of the eye exactly. Now the fact is, that even a slight displacement of the eye backward, forward, or laterally, makes a very considerable difference in the visible arc, especially as we approach the periphery. To center the globe we make use of the simple fact that the center of the arc of a circle like that of the perimeter is at the intersection of any two of its radii. Nearly all perimeters have an opening in the center of the arc through which the observer can look straight at the observed eye. It is, therefore, easy to place the eye on the line of that central radius.

But to place the observed eye in the line of some other radius requires more care. For this purpose a strip of brass, about 5 cm. long, is attached to the edge of "the carrier," which traverses the arc of every perimeter, and, being attached at right angles to the face of the carrier, this strip is, of course, in the line of a radius of the arc of the perimeter, no matter at what point the carrier is placed. If, now, one end of this strip be turned at right angles to its flat surface and be made pointed, and the other end also be turned in the same manner and have a small notch at the end, the whole strip forms what we ordinarily call a "gunsight," the line of this gunsight being in the line of a radius of the arc of the perimeter. That is to say, if the subject under examination is told to look through the opening which is in the center of the arc, the eye is in the line of the central radius of the arc of the perimeter. Then, if the surgeon sights along the line of this gunsight at the globe, and moves the head of the patient backward and forward until the line of the gunsight strikes near the edge of the observed cornea, evidently the

prolongation of that line along the gunsight would intersect also the center of the globe. In that position the center of the eye corresponds to the center of the arc of the perimeter.

There is a second way of making sure that this second radius passes through the center of the eye. If a small electric light is fixed below the gunsight, then the observed eye can be rotated so that the reflection on it, of that light, can be brought to the center of the cornea when the subject looks at the light, no matter from what point of the arc of the perimeter the observation is made. In that case, evidently, the center of the globe is at the center of that arc.

Having thus made the center of the observed eye correspond with the center of the arc of the perimeter, the next desideratum is to keep it in that place. To do that the head must be firmly fixed. The usual chin-rest is entirely insufficient, especially with a restless patient. But the head can be kept at rest if, instead of the chin-rest, we use the wooden bit which was first figured by Helmholtz in his "Physiologic Optics," and which has since been adapted to Stevens' tropometer and to the ophthalmic head-rest which I have once described. This head-rest can be fitted to almost any perimeter, and slides from one side to the other so as to be adapted for either eye. It needs no special description.

In a word, with the aid of the central opening of the perimeter and the "gunsight" the center of the observed eye can be brought to the center of the arc, and by means of the Helmholtz bit it can be kept firmly in that position.

The third desideratum for these measurements relates to the perception of the test-object. This evidently depends upon three factors:

1. The size of the object.
2. The intensity of its illumination, and—
3. Its color.

In spite of the importance of these three factors almost no attempt has been made to obtain uniformity by inventors or

by instrument-makers. The field of vision which is published by one observer may not be at all the same field which another would obtain with his perimeter from the same patient.

In deciding as to what shall be the size of the object or the intensity of the illumination, it is evidently desirable to select the minimum of each which can be recognized under normal conditions by different portions of the normal retina. This minimum has been determined by Fechner, Aubert, and several other investigators. It would lead to too long a digression to discuss in this connection this minimum of visible illumination, or what physiologists have called the threshold.

There are evident difficulties in attempting to produce this minimum illumination and in keeping it constant. The usual method of passing a white or colored object of indefinite size along the arc of a perimeter in daylight of indefinite illumination is a test without much scientific value. The source of illumination must be a very weak electric light in a perfectly dark room. Of course, the intensity of any lamp decreases with its use, and it is evidently impracticable to attempt frequent photometric measurements or comparisons of the test light with a given standard. The faintest glimmer of light which the normal eye of the examiner can perceive in a given portion of his own field of vision should be perceived, of course, by the patient if his field is also normal. The light which seems best practically is not more than one-fifth of a candle power, passing through an opening 1.5 mm. in diameter, and that too shaded by a protective covering.

The next point to be taken into consideration is the color of the light, as, for well-known reasons, we wish to obtain the field for the red and blue, as well as for the white light. The exact shade of the colored light is not of as great importance as the makers of some perimeters would have us believe. An important feature is that the light, of whatever intensity

or color which emanates from the object, shall not cause reflections on other parts of the perimeter or from objects in the room. Such reflections enable the patient to see with a portion of the retina which may be normal, whereas the surgeon thinks that the light falls only on a part of the retina which in reality is abnormal.

For this reason the room in which the examination is made should be as dark as the ophthalmoscope room. This darkness helps to shut off these extraneous reflections from objections in the room, and also gives the test light its proper brilliancy.

Reflections from the instrument itself can be obviated by placing a cylinder four or five centimeters long and one and a half centimeters in diameter around the light, and making the inside of this cylinder black to prevent reflections. As the axis of this cylinder is in the line of a radius of the perimeter, the light from the illuminated point falls straight on the opposite part of the retina, and is not diffused so as to cause the confusing reflections already referred to.

Another point to be taken into consideration is the method by which the test-object is called to the attention of the patient. This should be such as to eliminate, as far as possible, the element of suggestion by the examiner. The ordinary method of asking a patient if he can see a test-object "now," and again "now," and "now," is extremely unreliable, especially when dealing with ignorant or susceptible subjects. A much better method is to slide the test-object of whatever color it may be quietly along the arc, and to a certain point, and then, without saying a word or making a gesture, close the circuit and flash the light. The subject should then raise his finger or hand which has been laid on the table. If he attempts to say "no" or "yes," he is very apt to move his teeth from the Helmholtz bit and to disturb the position of the head.

Finally, a self-registering attachment similar to that first

suggested by McHardy is a very convenient, though not an essential, part of every perimeter.

All that has been said thus far refers to the use of the perimeter as an instrument for measuring the field of vision. But it is evident that the same appliances used in the same way are also valuable when the arc is used as a tropometer. It is out of place here to discuss the importance of the tropometer. It need only be stated that, with an arc of this construction, we cannot measure the arc of rotation inward on account of the prominence of the nose, nor downward because of the difficulty of the surgeon to sight over the arc from below upward. But when the globe is rotated in the horizontal direction outward, the extent of the arc can be measured with great exactness by sighting over the gunsight, and observing on the cornea the reflection of the test light. When the perimeter is used as a tropometer, of course the electric light must be much stronger, one of several candle power being necessary to show the corneal reflex distinctly even in a dark room.

It may be stated, by way of parentheses, that the extent of the arc of rotation is in itself a small indication of the motility of the globe in any direction. The important point is the *behavior* of the globe as it reaches the extreme limit of the rotation, for, if the globe can be rotated outward, say 55 or 60 degrees, in the horizontal plane and can be held stationary at that point without variation, or any "wabbling" motion, we may assume that the rotation outward is normal. On the other hand, when the globe reaches a point near its extreme limit of outward rotation, if it there halts, or jerks, or wabbles, we at once suspect an insufficient or paretic action of the abductors. For example, in a case of esotropia such a behavior of a globe would mean that the eye turns in, rather because the abductors are too weak than because the adductors are too strong, and that conclusion means, in such cases, an operation for advancement of the externus.

On the other hand, in a case of esotropia, if the rotation outward is normal and steady, it is probable that other measurements also would show that the esotropia is due more to excessive action of the adductors than to weakness of the abductors, and under such conditions any one of the different forms of tenotomy of the internal rectus would be apt to give a good result. This important point cannot be elaborated in this connection.

In a word, with the aid of appliances here described, we can increase greatly the exactness of measurements of the fields of vision and of arcs of rotation, and these improvements in diagnoses mean corresponding improvements in methods of treatment.

OCULAR PHENOMENA ACCOMPANYING THREE CASES OF GASTRO-INTESTINAL DISORDER.

HIRAM WOODS, M.D.,
Baltimore, Md.

The cases are as follows:

CASE I.—*Chronic Uveitis.*—Lawyer, aged forty-eight years, consulted me in 1910 for refraction correction, compound hyperopic astigmatism, and presbyopia. Eye-grounds normal. I did not see him again until April, 1913. For several days he had had hyperemia of the conjunctiva without secretion, limited to the left eye, which was slightly sensitive to touch. Six days later he showed left descemetitis, for which no explanation could be found. This condition persisted for seven months. Vision, usually normal, never fell below $\frac{20}{30}$, with unaffected form and color fields. There was occasional circumcorneal hyperemia. The pupil contracted sharply several times, but no synechia formed. The right eye remained normal. Late in November, while at court trial table, he became conscious of something wrong

in the left eye. Covering the right, he found the left was practically blind. The next day V. = $\frac{3}{200}$, and the media so cloudy that nothing could be seen. Descemetitis covered nearly the whole cornea. In two weeks vision had improved to $\frac{20}{200}$. During the winter of 1913–14 the eye remained in about this condition: persistent conjunctival hyperemia and descemetitis, vision varying from $\frac{20}{100}$ to $\frac{10}{200}$. In March, 1914, there developed, in the space of two or three days, posterior synechia, which yielded slowly to atropin. The right eye had remained free of trouble during the year of inflammation in the left. April 22, 1914, there was right descemetitis. The left eye at this time showed a hazy vitreous with muscæ, but I could find neither fundal lesion nor scotoma. V. R. E. = $\frac{20}{20}$; L. E. = $\frac{15}{200}$. From this time until July 15th the only change was that vision improved in the right to $\frac{20}{15}$; left, $\frac{20}{50}$ to $\frac{20}{70}$. From September 30th to January, 1915, the conjunctival hyperemia and descemetitis persisted in both eyes, with sudden transient attacks of increased vitreous clouding, with reduction of vision during October and November.

On January 16, 1915, a large upper left molar "dead tooth," which had been crowned several years previously and had become painful, was extracted. A drop or two of pus followed. Two days later both eyes became violently red, and vision fell in the right eye from $\frac{20}{15}$ to $\frac{20}{100}$, and in the left eye to $\frac{5}{200}$. The vitreous of each eye was so cloudy I could not see the fundus. That afternoon he had to be led into the hospital. Under diaphoresis, etc., he gradually improved. His condition June, 1915, is chronic conjunctival hyperemia and descemetitis in both eyes; V. R. E. = $\frac{20}{15}$; L. E. = $\frac{20}{20}$.

This is the eye history for a little over two years. Turning now to efforts to determine etiology, I have long since looked upon conjunctival congestion without secretion as a harbinger of mischief. It is seen as a first symptom of recurrent iritis, episcleritis, and uveal inflammation. It sometimes comes and goes without explanation, though the more carefully we inspect the teeth, look for nasal troubles, and inquire into what we vaguely call digestive disturbances, the fewer become "idiopathic" cases. There was no reason to suspect lues or tuberculosis. Blood and clinical examinations, however, excluded both. There was no history of

recent acute illness. The crowned dead molar excited my suspicion from the beginning, but it was giving no pain, and the dentist pronounced it free from disease. Learning that the patient had been under the care of Dr. William I. Messick, Associate in Gastro-intestinal Diseases at the University of Maryland, I asked the doctor to join me in attendance.

Some ten years or so before the eye trouble Dr. Messick had been consulted for acid eructations, constipation, and a train of disturbances which had been attributed to the gastro-intestinal canal. These so-called toxic evidences were closely connected with the mucous membrane of the nose and eye. Swelling of the turbinals and recurrent conjunctival redness had been observed over a period of years. Dr. Messick thought that he had always been able to trace these things, at least coincidentally, to errors in diet—rather overeating than partaking of any particular article of food. Be this as it may, there had been found great variation in the hydrochloric acid content—usually a hyperacidity. Indican was frequently found in excess, and again would disappear. Undigested meat was found in the stools. Obstinate constipation usually followed free eating of meats, and at all times was hard to prevent. During the course of uveitis, with sudden depressions in vision, Dr. Messick found the same association observed through previous years. For instance, the sudden failure in vision in November, 1914, was preceded by free indulgence in eating, with characteristic findings in urine and stools. Finally there came the sudden collapse of both eyes after the extraction of the crowned tooth, with evacuation of pus. Before this, systematic physical exercise and regulation of diet had partially remedied the constipation habit. The flare-up of uveitis when the alveolar pus-sac was brought into contact with freshly opened tissue seems to indicate an immediate absorption, the toxins, or whatever the offending substance, going directly to the eyes. Dr. Thomas R. Brown and Dr. William S. Thayer saw the patient after the tooth experience. Dr. Brown's examination showed achylia, although the tendency to hyperacidity in Dr. Messick's previous test was pronounced, and the condition returned later. Dr. Brown has told me he thinks it quite possible that absorption from

the alveolar pus-sac could produce all the varying intestinal symptoms and the ocular inflammation. Dr. Thayer wrote me: "The relation of the ocular attacks to the attention of the teeth seems to me extremely significant. He has a good deal of pyorrhea. . . . As one considers the whole situation, I should be inclined to lay more stress on the teeth than on anything else. . . . Very often in arthritis one finds a direct reaction after interference with the inflammatory focus, an alveolar abscess, or the scraping of the gums in pyorrhea at the beginning of the treatment, when there is still a great deal of pus, and my feeling would be that he ought to have those gums washed every month for a while; they ought to be kept absolutely clean after the immediate attention that is now being given to those teeth which were particularly affected."

Improvement has been steady since January of this year, when the tooth was extracted. The eyes are still red, and descemetitis persists. Vision is normal in both eyes. Physical exercise and abdominal massage are getting the credit for improvement in constipation, may be justly.

Case II.—*Paracentral Color Scotoma.*—Dr. X, aged forty-nine years, first consulted me in February, 1906, for refraction correction. Vision was normal in each eye. Fields were not taken. Four years prior to this consultation Dr. I. E. Atkinson, now deceased, had suspected commencing chronic nephritis. Urinalysis failed, however, to show anything suggestive of this condition. I did not see Dr. X again professionally for six years (March, 1912), when he came because vision was poor. Tests at various times during four days gave vision from normal to one-half or less. Under euphthalmin dilatation I could find nothing abnormal. In each eye there were contracted form fields, with enlarged red scotoma extending from Mariotte's spot to the fovea (Fig. 1). The form fields at two successive examinations are shown in Fig. 1; the inner line, March 14th; outer, April 8th. Limitations of color perception are not recorded, but they did not extend over 10 or 15 degrees from the fixation-point.

Dr. X is a very large man, weighing at the time of examination nearly 300 pounds. He used neither tobacco nor alcohol. Urinalysis showed nothing except indican, appar-

ently in excessive quantity. The fecal odor of the breath was decided. He was sent to the University Hospital and put under Dr. Messick's care. His report, embracing, as it does, an illness just prior to the patient's coming to me, is as follows: "A month before coming to Baltimore, while on his way east from Indiana, the patient was taken ill on the train with severe gastric pain. He was ill for two days in a hospital in Pittsburgh. There had been no recurrence of these acute symptoms, but there were loss of appetite, constipation, a fecal breath, and gingivitis. The urine at first showed nothing except indican. During the month that Dr. X was at the University Hospital there was occasionally glycosuria,

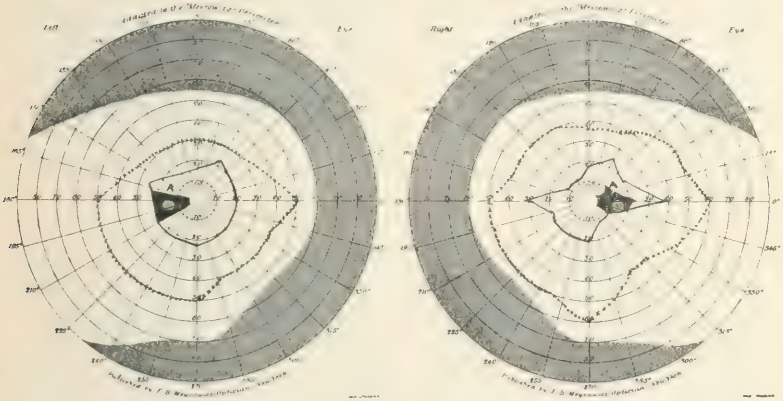


Fig. 1.—Form fields at two successive examinations. A, Paracentral scotoma for red.

which would disappear over night. At no time could albumin or casts be found. There was marked gastric hyperacidity. Stools showed muscle-fiber, free fat, and fatty acids. Treatment consisted of antacid diet, purgations, etc., and for a time he seemed to improve." (It was then that the improvement in form field was noted, as shown in Fig. 1.)

Severe pain returning toward the middle of April, the man was referred to Dr. Frank Martin. Radiographs revealed an almost complete obstruction of the transverse colon near the splenic flexure. Operation traced this to a band of fat around the colon, and revealed gall-stones, which had never been suspected. These were removed. Recovery from

operation was uninterrupted, and two months later central vision was normal, but with practically no change in the field limits. However, the red scotoma had disappeared from the right eye. Up to July 7, 1914, it had remained in the left. After operation the patient remained well until May, 1913, when he had an attack of abdominal pain, with swelling in the right hypochondrium. The eyes were not affected. Dr. Martin opened an abscess communicating with the gall-bladder.

Dr. X was not seen again professionally until February, 1915, when he came with blurred sight, and examination showed V. R. E. = $\frac{20}{100}$; L. E. = $\frac{20}{30}$. In the right fundus there were several spots of exudate above the fovea. Traces of sugar were found in the urine. We have found glycosuria occasionally for two years. It has usually been after indulgence in starchy food. Only in February last did the eyes show trouble at such periods. Within a week, and after free purgation, rest, regulation of diet, etc., the spots disappeared from the right retina and sugar from the urine. Vision became normal.

Case III.—*Shifting Toxic (?) Scotoma.*—Man, aged forty-two years, consulted me April 8, 1913. He is head of a corporation and carries large responsibilities. His habits are similar to those of many successful business men: works hard while he is at it, sacrifices leisure to business, if necessary, and indulges sparingly in tobacco and alcohol, often going for weeks without the latter. His business compels him "to frequently play the rôle of entertainer," to use his own phrase, and he says it is usually after these, not always voluntary, experiences that he has the visual changes to be narrated. His general health has always been excellent. In 1901 he underwent an operation for acute appendicitis, but this is his only illness. His first eye trouble occurred about fifteen years ago. He was in Atlantic City, N. J., and consulted Dr. Pollard. The trouble was confined to the left eye, and he says that Dr. Pollard told him he would not hesitate to attribute his visual defects to the excessive use of alcohol or tobacco if he did not know he was addicted to neither. The patient described quite accurately a left paracentral scotoma. This was easily demonstrated by my first

examination, and is shown in Fig. 2. Near the area of lost perception there is another where the white disc is perceived only dimly. This condition runs through several fields, the areas of blindness and dim vision changing in shape and

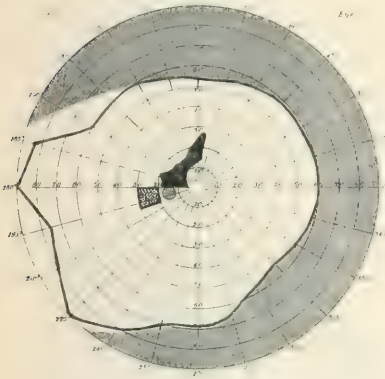


Fig. 2.—Field April 8, 1913.

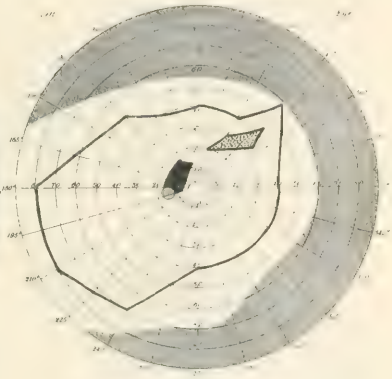


Fig. 3.—Field June 4, 1913.

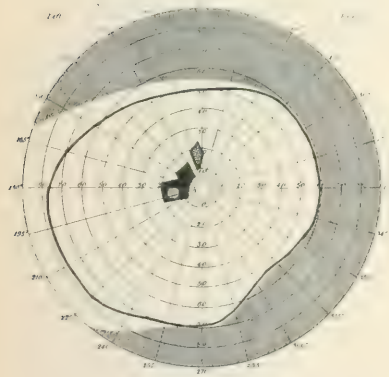


Fig. 4.—Field October 5, 1913.

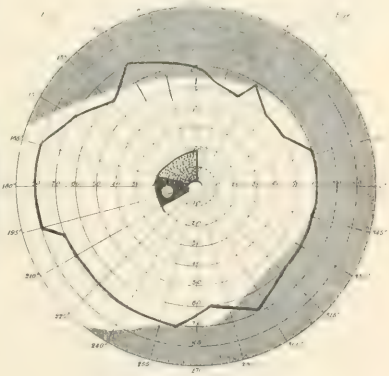


Fig. 5.—Field May 7, 1914.

shifting in position. Figs. 2 to 5 show this at different dates. Intermediate fields are omitted.

The reason for his consulting me was that he had noticed, the day before, a dim area in the right, similar to that in the left, eye which had taken him to Dr. Pollard years ago.

Ophthalmoscopic appearance was negative. V. R. E. = $\frac{20}{40}$; sph. + 1.50 = $\frac{20}{24}$; left = $\frac{20}{30}$, with the same H. Right field is shown in Fig. 6, and has the general characteristics of that of the left eye. There was an absolute scotoma below the fovea, with an adjacent area of dim perception in the lower temporal field.

Three days before he saw me he went to an after-theater party, where he drank, he says, not more than two glasses of light wine. He never drinks whisky. Next day he was exposed to a cold wind while motoring, and was conscious of pain in his right temple. I thought the case one of acute retrobulbar neuritis, but whether due to unusual suscepti-

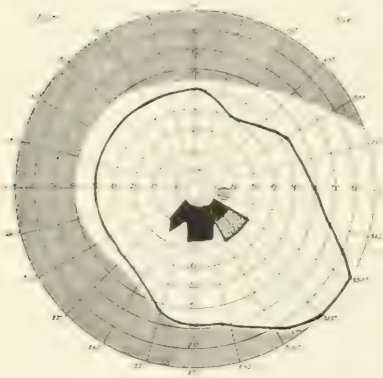


Fig. 6.—Field April 8, 1913.

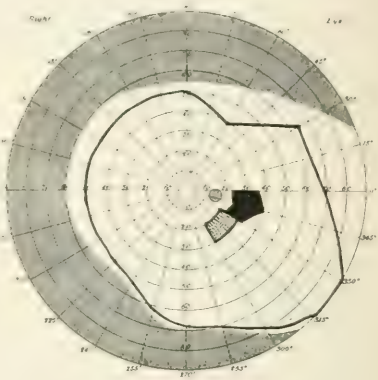


Fig. 7.—Field May 8, 1913.

bility to alcohol or to exposure I could not say. However, he was given purgation, and told to abstain absolutely from alcohol and tobacco. One month from the date of his first visit he had the right field depicted in Fig. 7. It will be noted that the absolute scotoma, with adjacent dim area, has shifted into the temporal field. Fig. 8, taken one week later, shows no absolute blindness, but the area of dim vision has shifted to the fovea and perifoveal region. Fig. 9, taken about four weeks later, shows a clear foveal region, but with the return of the dim area in the lower and outer temporal quadrant. The fields were taken with a 5 mm. disc. I was careful to eliminate change of position, and think I succeeded.

On October 5, 1913, after a fall from grace in the shape of a dinner, his right field was as in Fig. 10. Central vision was $\frac{20}{20}$, but there was an absolute scotoma in the nasal field and an area of dim perception in the temporal. On May 7, 1914,

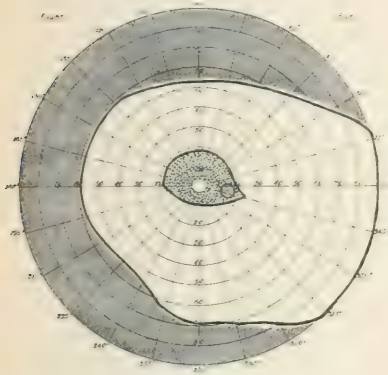


Fig. 8.—Field May 16, 1913.

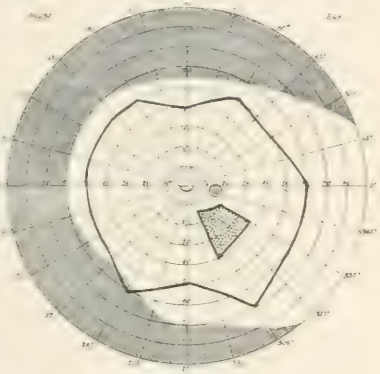


Fig. 9.—Field June 4, 1913.

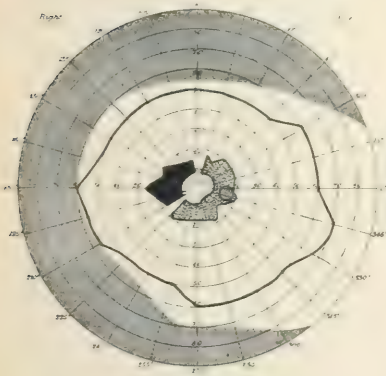


Fig. 10.—Field October 5, 1913.

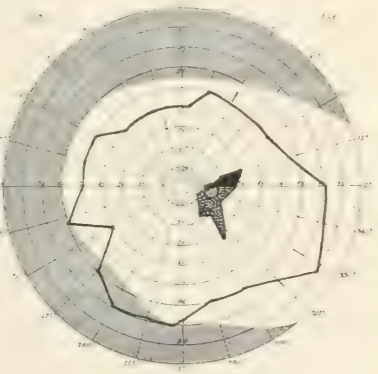


Fig. 11.—Field May 7, 1914.

after a similar dietetic experience, the right eye presented the field shown in Fig. 11. There is an area of absolute blindness in the temporal field, just above Mariotte's spot, and dim below.

I have omitted the lines for color because they merely

varied with the form limits. Of course, they could not be noted in the scotomatous areas.

The patient was put under Dr. Messick's care soon after I saw him in 1913. Dr. Messick reported gaseous eructations, constipation, indicanuria in excess, and functional gastric hyperacidity. A striking feature about the case is that the blood-pressure has never been higher than 95 mm. measured on the Tycos instrument. I did not see the patient professionally after the summer of 1914 until I asked him to come to my office on June 21, 1915. He told me he had been entirely well and free from eye attacks for a year; that the restricted diet ordered by Dr. Messick was not enough to keep up his nutrition, and he had consulted Dr. Thomas R. Brown, who confirmed Dr. Messick's findings, but allowed him a freer diet. The patient himself says that he attributes his freedom from eye attacks during the past year not to any virtuous abstinence from an occasional glass of wine or cigars, but to his self-prescribed daily use of a purgative water. Be this as it may, he is in excellent health; right eye has $\frac{20}{15}$ vision, without defect in either form or color fields. The left has $\frac{20}{20}$, with a negative scotoma in the upper temporal field adjacent to Mariotte's spot. His limits for blue and red are about 5 and 10 degrees inside a slightly contracted form line.

These three cases have one feature in common—classic symptoms of intestinal stasis, with remote troubles rightly or wrongly attributed to the intestinal condition. The remote effect in case one was a chronic uveitis and the ultimate result indicates that, for nearly two years, there was an unrecognized focus of infection in the alveolar process. The case was submitted to every means of diagnosis except an *x*-ray picture. The primary lesson is that, to the multitude of councilors the oculist is forced to call in to help in a diagnosis of the cause of uveitis, must often be added the *x*-ray expert. Again, the numerous relapses in visual acuity were always accompanied by evidence of renewed intestinal disturbances, so one cannot resist the suspicion that the alveolar focus may have been a later development; true, the

cause of the final outbreak in January, 1915, but not the original cause of uveitis two years previous, one cannot say definitely. I think, however, that Dr. Thayer is right in attributing to the teeth the chief causative factor. This belief is confirmed by Dr. Brown's opinion that absorption from an alveolar process might have produced many features of the intestinal disorders. De Schweinitz, in his London address of 1913 upon the "Pathogenesis of Chronic Uveitis, Excluding the Syphilitic, Tuberculous, and Sympathetic Varieties," quotes Harold Sims to the same effect, as follows:

"Doubtless in many patients with pyorrhea proper investigations will reveal indicanuria, acidosis, and, in general terms, the usual signs of so-called autointoxication, and while treatment to relieve the perverted metabolism may be of value, there is no evidence of a satisfying character that the acidosis, etc., are the cause of the septic mouth condition. It is more likely that oral sepsis originates the autoinfection as well as the local inflammatory processes in joints and uveal tract tissues."

The second case shows usual laboratory symptoms of intestinal stasis, with eye complications quite characteristic of something else—toxic amblyopia, usually from tobacco or alcohol. Yet the patient used neither. Further evidence of infection is found in the gall-stones and subsequent abscess, while occasional glycosuria emphasizes, if nothing else, a tendency to a carbohydrate intolerance. The case seems to show the possibility of a distant infection producing, probably in the optic nerve, the same functional disability which characterizes the intake of certain toxic substances. It opens up the whole question of formation of toxins following infection—a doctrine ably presented and defended by Alex. C. Abbott, in de Schweinitz's paper. Again, the sudden failure of vision in the right eye this past winter was accompanied by exudates in the retina. At the same time there was transient glycosuria, and laboratory examination by Dr.

Messick gave positive evidence of what we call intestinal autointoxication. Within a few days, with clearing of intestinal conditions, the spots disappeared. Have we not here a condition like that presented in the retinitis of pregnancy (wrongly, I think, called albuminuric because of a coexisting albuminuria), and disappearing as soon as the uterus is emptied? At the same time the kidneys return to normal unless there is a preëxisting nephritis. And are we not forced to accept as the most likely cause a toxemia from the intestines, finding in the eye its "combining affinity," to use Abbott's expression, be it the result of chemic changes, as is very improbable, or the holding up of bacteria in the intestines, as is generally believed.

The chief teaching of the case is the connection between undoubted infection situated in the intestinal canal and eye symptoms which must be explained on the basis of a toxemia, if explained at all. Their transient nature seems to exclude other causes. As to the third case, I have never seen anything like these shifting scotomata. The only analogy I know of is the migraine scotomata, which are so hard to record because of their short existence. At one time I was disposed to regard the attacks as a sort of abortive migraine, but this became evidently absurd. Arterial spasm suggests itself; even so, we do not know the cause. The clear relationship between these occurrences and the intake of anything alcoholic was too apparent to be disregarded. Freedom during the past year, when intestinal stasis seems to have been overcome, tends to support Horner's contention regarding the relationship of alcohol and tobacco amblyopia to intestinal disorders: that the presence of the latter is necessary to make the former effective in producing the chemical agent, or whatever it is, which affects the optic nerve, probably causing a local anemia.

What we call "remote results of perverted metabolism" is a far-reaching problem. My friend, Dr. Thomas R. Brown,

with whom I have seen several cases, has allowed me to use the manuscript of a paper read during the past winter before the Pittsburgh Academy of Medicine, and now published in the *Pennsylvania Medical Journal*, June, 1915. The title is: "The Medical Aspect of Intestinal Stasis," which is defined as "the result of atony of the intestines and abdominal muscles allowing the viscera to prolapse, with tendency to kinking of bowels, and with the possibility of the content staying far beyond the normal limit at or above the involved area." The local results are:

"A stasis of fecal contents, generally associated with constipation, but sometimes with apparent diarrhea, the possibility of changes in mucosa and musculature, of the formations of erosions and ulcerations, the development of a low grade of peritonitis, a more and more sluggish peristalsis, with longer and longer stay of fecal column, an increased permeability of the intestinal wall to bacteria and to other substances, a loss of sphincter action so necessary to fecal propulsion, the development of adhesions, deleterious influences on the local secretions, reflex disturbances in the chemical, sensory, or motor spheres of other portions of the digestive tract or even outside the digestive apparatus; disturbances of the nervous mechanism by pressure, by traction, and possibly by the influences of toxins, change in the cultural conditions, with the possibility of the appearance of microörganisms not found in conditions of health or of overgrowth of certain bacteria normally present in minimal amount, with diminution of certain of the strains normally found. . . . Careful studies of the stool, and quantitative estimation of the diastatic and tryptic ferments therein, have made us feel that functional or even organic disturbances of the pancreas may be met with in certain of these cases, also suggested by the temporary glycosuria met with in certain of the acute toxic outbreaks. The skin lesions are peculiarly interesting, because of the isolation, by Eppinger, from the stool in certain cases of autointoxication, of a ptomain, which even in great dilution produced marked urticaria if applied locally.

"After presenting local and distant symptoms of stasis, the writer reviews indican and the ethereal sulphates, and concludes that they have not much significance. Regarding the theoretic explanations of distant effects, he concludes that autointoxication is at present the best solution. As to the nature of this intoxication, he asks the following: Is it a toxin normally present but in excess? Or, due to this stasis, have the liver and other portions of the protective mechanism become insufficient (possibly due to repeated mild anaphylactic reactions from proteid poisoning, as suggested by Longcope's recent work), and toxins normally destroyed by the liver allowed to get into the general circulation? Or is there a tendency in these cases for an overgrowth of certain forms of bacteria? Or is the toxemia due to the products of certain ferments in excessive amount or perverted? . . . There must be a breaking-down of the natural protective mechanism of the body. In the first place, the intestinal mucous membrane; in the second place, the liver; in the third place, certain glands of internal secretion, notably the thyroid, suprarenal body, and pituitary gland. . . . If these protective organs and tissues do not function properly, then the toxins reach the general circulation, from which they may be eliminated in the respired air, the urine, the stool, the sweat, possibly producing functional disturbances or permanent lesions in their excretion. If this excretion is not sufficient; if the body as a whole or certain organs or tissues have a lowered resistance or increased susceptibility, we then have the picture of autointoxication—in the one case producing a picture of psychoneurasthenia, in another confining its symptomatology largely to the gastro-intestinal tract, and possibly affecting some portion far removed from the primary lesion, and in still another group of cases affecting one organ alone, either due to its lowered resistance or its increased susceptibility, or possibly to a definite specificity of toxin.

"That the poisons cannot be demonstrated is not so much of a proof of their absence as of their extreme complexity, and of the minimal amounts in which they must be present in the blood, urine, and stool. . . .

"From the facts presented in the foregoing paper can we reconstruct the picture of intestinal stasis and the symptoms

it produces? If we depend solely upon proved phenomena, this is not possible—there would be too many gaps. The image would be blurred in many of its salient features. But if we believe, with Brieger, that 'our researches in the domain of physiologic chemistry are as yet so incomplete, so insufficient, that a negative result proves nothing at all,' and with Eppinger, that persistent research may lead to the isolation of many of the intestinal toxins, and feel that a clean-cut picture on definite possibilities is better than the indistinct image which must come if only proved facts are accepted, then I think we are justified in trying to reconstruct and visualize the symptom complex of intestinal stasis."

This extended quotation shows two things: First, all explanations are hypothetic; secondly, the problem involves knowledge of structure and functions of which we know very little. When we attempt to trace a path from an intestinal infection, even admitting the existence of toxins, to perverted eye function or lesion, we are without a solid basis for reasoning. There remain only observed clinical facts; yet these facts are too suggestive to allow dismissal.

DISCUSSION.

DR. C. H. MAY, New York: The paper of Dr. Woods reminds me of a patient who was sent to the Mt. Sinai Hospital for treatment, the principal symptom being an absolute central scotoma of one eye. When the condition was at its worst, it extended 10 degrees from the point of fixation. There was no evident reason for the presence of this condition. The vision was $\frac{20}{50}$. The patient was subjected to all kinds of tests, and it was finally concluded that the trouble was due to intestinal intoxication. After cathartics had been used liberally, and high irrigation, the vision would increase and the scotoma would become relative, extending but two or three degrees from the fixation-point. Then we proved that the eye condition was dependent upon the state of the gastro-intestinal tract: He was allowed to become constipated for several days, and the field of vision was then taken. The central scotoma was found absolute 10 degrees

from the point of fixation, and the vision reduced to $\frac{20}{200}$ or $\frac{20}{70}$. A number of experiments were made, and each time the result was the same. With constipation, there was an increase in the size of the scotoma and a reduction of vision. When the bowels were cleared out and high colon irrigation made use of, the vision improved and the central scotoma, although it did not disappear entirely, became relative and smaller. The man was kept under observation for two years, and during this time there were attacks of the same character, subject to the same changes, according to whether the bowels had been properly emptied or not.

Dr. W. Gordon M. Byers, Montreal, Canada: I should like to ask whether Dr. Woods would entertain the idea that in his second case the axial neuritis was due to the accompanying glycosuria. In the two cases of this kind that I have seen the amount of sugar present was very small indeed.

Dr. Hiram Woods, Baltimore, Md.: In answer to Dr. Byers, the glycosuria was transitory and recurrent in nature. It was never found when he was kept under the condition Dr. May has described in his patient; that is, when his bowels were kept free and he had no digestive disturbance. He also had high colon irrigations at times. Transient glycosuria is studied in a paper by Dr. Thomas R. Brown, from which I quote in my own paper. Dr. Brown thinks transient glycosuria may be due to secondary functional involvement of the pancreas. Again, the disappearance of the retinal spots argues against a diabetic cause. The author reviews various hypothetic explanations of remote symptoms of abdominal stasis, and finally admits that none can be proved. He thinks the conditions are probably due to a toxin, and that the failure to discover this toxin is the result of the minimal amount in which it is present, and inadequate means of investigation. He believes a toxemia, based on retention and putrefaction, is the best explanation we have.

In the paper by Dr. de Schweinitz, mentioned in my paper, there is quoted a lengthy conversation with Dr. Abbott, in which the latter takes the same ground—we have to admit the presence of a toxin in order to understand the

existence of a great many things. He thinks that it is present in the eye, and attacks tissues for which it has a "chemical affinity"; that the condition produced is clinically the same as an inflammation, without being due to bacteria. Function is destroyed.

LUMBAR PUNCTURE AND EXAMINATION OF THE SPINAL FLUID IN AFFECTIONS OF THE EYE.*

JAMES B. AYER, M.D.,
Boston, Mass.

It is my privilege to present the results of examination of the spinal fluid in a group of cases of common interest to neurologist and ophthalmologist, together with effects of treatment in certain of the syphilitic ones. The cases have, for the most part, been seen both in the Nerve Room of the Massachusetts General Hospital and at the Massachusetts Eye and Ear Infirmary; to these have been added private cases, many of which have been referred to me, first or second hand, by ophthalmologists.

The recognition of the value of the examination of the spinal fluid has steadily increased, and its sphere of importance has gradually enlarged since its inception by Quincke, twenty years ago. At first employed almost entirely in cases of acute meningitis, it was later found that chronic inflammatory changes in the nervous system could be diagnosticated also, and later still it became possible, by this means, to differentiate the large and important group of nerve syphilis from other conditions. Nor has the spinal route been used solely for diagnostic purposes, for, as is well known, some of the most satisfactory medicaments are now

* Presented by invitation.

given into the subdural space. Our problem to-day is to differentiate more carefully, by means of the spinal fluid, types of inflammatory and degenerative disease of the nervous system; and this method of investigation should be of the utmost interest to the eye specialist, in that he alone so often has the first actual view of the nervous system.

While the inflammatory conditions are the most readily diagnosticated to-day, much light may be thrown upon other conditions from examination of the spinal fluid, *e. g.*, as to the nature of cerebral hypertension, and we may, I believe, confidently look to the future for aid in diagnosis of other conditions, particularly those disorders of internal secretions, interrelated, but ill understood.

The paper is based on the examination of 160 cases of concern to the ophthalmologist, with a background of a much larger number of less special interest. As has been hinted at, our problem in the greater number of cases was simply, Is this case syphilitic or not? In some the examination was intended to be only confirmatory, or for the sake of following the effect of treatment; in others the diagnosis depended upon the findings, and, if I may anticipate a little, I believe that the greatest value of puncture is to be found not in examination of an out-and-out case of tabes or evidently syphilitic optic atrophy (this is like using a thermometer in a clear case of fever), but its use in a case of slightly unequal or irregular pupils, perhaps with sluggish reaction, in early diplopia, with or without evident palsy, suspected beginning atrophy, and of certain forms of headache, epilepsy, and migraine. In these latter a syphilitic process will often show in the spinal fluid even more conclusively than in the later stages, though evident then at a glance. Let us then take up in turn the various groups of cases in which we have examined the spinal fluid, and see what we can learn from them; and first the large group of external oculomotor

palsies of all kinds, partial and complete, for the most part of some permanence, and not transitory.

EXTERNAL OCULAR PALSIES.

There were 40 cases of this character. In many the tests were performed as one extra link in the diagnostic chain of tabes, but in at least 11 the tests were of prime importance in the diagnosis of the case, the oculomotor palsy being the sole sign. A typical example of such a case is illustrated by K. C., aged forty-seven years, who awakened one morning with the right eye closed. Examination showed only third-nerve palsy; blood test gave a negative Wassermann, but the spinal fluid gave a strongly positive reaction and a goldsol reaction in the syphilitic zone. Not only is the examination of confirmatory value, however, where you suspect syphilis. In the case of Mrs. A. G., all examiners diagnosticated cerebral syphilis, and the spinal fluid seemed to confirm this in that there was increased number of cells, though the Wassermann was negative. Salvarsan was freely given, but the patient continuously grew worse, so that other cranial nerves became involved, and later one side of the body; a mass appeared in the ethmoid region which showed carcinoma, and our diagnosis had to be changed to tumor, thereby agreeing with the dictates of the spinal fluid examination.

Of the 40 cases, all except 5 show evidence of meningeal irritation, as demonstrated by increase of number of cells per centimeter, increased proteids, a positive Wassermann reaction or gold chlorid test, and usually more than one. That is, only 5 cases show normal spinal fluids; of these, one was really a case of subjective diplopia and has been helped by psychotherapy; another has signs indicative of nerve syphilis, but these signs were the same in 1915 as in 1908, and he has no symptoms suggestive of a progressive process. In the case of two others no cause has been found, yet no

other signs of syphilis have been noted. In the fifth case the spinal fluid was normal and the blood showed a positive Wassermann. This was the case in a young girl, recently married, and probably denotes a manifestation of acute vascular syphilis rather than of cerebrospinal infection.

Five others, while showing pathologic fluids, fail to show the specific reaction for syphilis. Three are evident cases of tabes, but the tests were done before the days of the reenforced antigen; the fourth case was that of cerebral tumor above mentioned, and the fifth has been considered polioencephalitis.

To summarize the cases of oculomotor palsy: In those not congenital or with other obvious cause usually a manifestation of syphilis of the nervous system. If all tests, blood and spinal fluid, are absolutely negative, the case is probably not syphilis, or represents a run-out process, the signs of which should be evident. In our experience oculomotor palsies due to an active syphilitic process are revealed by both positive blood and spinal fluid tests; occasionally the former is more reliable, but more often the latter.

Primary Optic Atrophy.

In 44 cases of primary optic atrophy we find only 12 spinal fluids with normal findings, but in four of these the tests were performed after antisyphilitic treatment, the blood having shown previously a positive Wassermann. Of the six cases where blood and spinal fluid both were negative, three were hardly thought to have been positive, for in one case tobacco played a large part; in another lead was demonstrated, and in a third there was a distinct family amaurotic tendency. A fourth exhibited atrophy of the superior half of her optic disc only, a condition not resembling the syphilitic atrophies. In two of this group were we surprised in our findings. Mr. G. M. had the usual signs of well-marked tabes, including Argyll-Robertson pupils;

he had, however, had no symptoms referable to this disease or any other disorder until, a short time before being seen, he had suddenly lost the sight in one eye. In the routine of an out-patient examination he was naturally set down as tabes with optic atrophy, with the usual bad prognosis. The report of the examination of blood and spinal fluid being absolutely negative, made us reconsider the history, and the following opinion of the patient seems a fairer one: The man had had tabes, leaving him with the signs of the disease, but, as there were none of the usual symptoms, and in view of the negative tests, it seemed fair to consider the pathologic process as quiescent. He has evidence of arteriosclerosis, with high blood-pressure; the optic atrophy was one-sided and sudden in onset, probably a vascular cause for this quite unlike the slow progress of a tabetic atrophy. We have evidence of syphilis as the probable cause of the arteriosclerosis. What, then, will be our prognosis after having performed the tests? Not a progressive, crippling, blinding disease, as we had expected, but a condition which might remain unchanged for many a year. We can, I think, safely assure him that the other eye is in no more danger than is any other part of the body in the presence of arteriosclerosis, and our treatment will be prophylactic in nature, rather than heroically antisyphilitic. This case is cited at length in order to give an idea of the reasoning which seems fair when all the facts are known, and the last fact, and sometimes the most important, is the examination of the spinal fluid. I shall watch with interest the progress of this patient, and so far—eight months—I am pleased to say there is no progress.

In another patient the typical reactions were expected which would confirm a diagnosis of tabetic optic atrophy. My suspicions were aroused that all was not as expected as soon as the canal was entered, for the spinal fluid mounted higher and higher into the manometer, until the alarming

height of 360 mm. had been reached. I was not comfortable that night, nor was the patient, as he gasped for breath, with pulse in the fifties, and it was not necessary to have a negative Wassermann and excessive protein content of the spinal fluid to tell me that we were dealing with a case of brain tumor. In reference to this subject I may say that, in cases of heightened intracranial pressure, especially where there is a subtentorial tumor, avoid lumbar puncture, or do it only when there is much to gain, and then do it with your eyes open. I have had two other scares besides the above-mentioned one, and could show you the flattened medulla of one patient dying of respiratory and cardiac paralysis the day following lumbar puncture. The best contraindication of puncture is perhaps the presence of well-marked choked disc.

The optic atrophies (so-called "primary") which have come to my hand appear then to be mostly associated with a true nerve syphilis, as evidenced by a pathologic spinal fluid of syphilitic nature. As in the case of oculomotor palsies, the blood usually runs parallel with the spinal fluid, but if I could have only one test, I should unhesitatingly choose the spinal. Normal tests, especially in cases where the atrophy has come on suddenly, are very strong evidence against its specific nature.

Concerning Treatment.—In the cases which show genuine involvement of the cerebrospinal system (by far the greater number), we are dealing with a chronic progressive disease, usually of many years' standing, which exhibits a pathology both of exudation and of degeneration. Our task is considerable, namely, to stop the pathologic process. This result is, I believe, accomplished in some, but in many more we can only help symptomatically, and arrest the disease temporarily or partially. It is, as a rule, easy to overcome an oculomotor palsy if taken early by various medicaments,

and, of course, these frequently disappear spontaneously—in one case a ptosis disappeared on withdrawal of a few cubic centimeters of spinal fluid. We have found, however, that the time-limit of such oculomotor palsy must not be over six months to obtain a complete recovery. As the paralysis is only one manifestation of a deep underlying process, our treatment must not stop at this—in fact, our task has only just begun. We must persist until not only symptoms are gone, but the spinal fluid has been reduced to normal, if possible.

As for the optic atrophies of specific origin: I am not encouraged about them. Many were treated by all means at our disposal, without arrest of the process. If taken when far advanced, I believe the outlook absolutely bad; if taken early, and the process is proceeding slowly, there is a chance that salvarsan will help.

Optic Neuritis and Choked Disc.

As has been said before, I am chary of tapping patients with choked disc. We have, however, 10 cases on whom lumbar puncture was performed at the time of the acute process, five of whom were shown to be syphilitic; we have, moreover, three other cases in whom the blood showed syphilis, the spinal fluid not being tested. As the blood Wassermann almost always runs parallel with the spinal fluid findings in these cases, on account of the possibility of increased intracranial pressure, it seems to me unwise to perform puncture in these cases unless there is some very good indication; the blood will usually be sufficient guide.

In contradistinction to the two preceding groups, it is gratifying to see the rapidity with which these cases, of syphilitic origin, clear with salvarsan. A. C., aged twenty-five years, one year after contracting syphilis had a prolonged attack of unconsciousness, and six months later, when seen by me, had choking of both discs. Tests positive for

syphilis, with evidence of a marked meningeal irritation, as shown by 110 cells per c.mm. After three doses of salvarsan of 0.3 gm. each, the fundi were of normal appearance. In six months more his fluid had been reduced to nearly normal. I have followed him for two years since then, and there has been no return of optic neuritis or any symptom traceable to syphilis: his blood and spinal fluid have remained essentially normal during this time. V. Z., aged twenty-eight years, complained of failing vision for eight months. He showed marked injection of the discs, concentric contraction of the fields, and pupils which reacted neither to light nor to accommodation. Tests were all positive for syphilis, the spinal fluid especially strong. It took four months of salvarsan treatment (eight injections intravenously and three intraspinally) before his discs were clear, and six months later his pupils reacted to accommodation. One year later, without more treatment, his vision was $\frac{20}{30}$ and $\frac{20}{20}$, his fundi, clear, but his pupils still of the Argyll-Robertson type.

Very different is the case of E. C. B., who gave negative Wassermann tests, but whose cell count remained high in spite of seven doses of salvarsan, and whose eyes went on to atrophy despite treatment. We have since been forced to abandon our diagnosis of syphilis in his case for that of tumor. One case, giving essentially negative findings, was treated, on the chances of being syphilis, with iodid and mercury, with rapid improvement, which has remained after three years. The tests, it is fair to say, done in 1912, were performed with the weaker reagent then in use.

In this group of optic neuritis, then, we have a right to rely upon our tests in differentiating syphilitic from non-syphilitic cases, and, for the most part, fortunately, the blood Wassermann is sufficient. Treatment, too, with older as with the newer syphilitic remedies is most gratifying, giving permanent improvement in symptoms and reduction of the pathologic process to normal.

ABNORMAL PUPILS.

To the writer the most interesting and the most valuable part of this subject centers about the abnormal pupil. General practitioners and specialists in all fields are or should be deeply interested in the study of the pupil. Of all manifestations of central nervous syphilis, the abnormal pupil is apt to be one of the earliest. But its changes are so various and often so slight that the physician is likely to be, on the one hand, too credulous, or, on the other hand, to overlook significant variations from the normal. Fortunately, the newer laboratory tests may with profit be used to determine in a given case whether or not a "specific pupil" indicates active syphilis or not. First let us consider the most important group:

The Argyll-Robertson Pupil.—This sign is generally considered as indicative of nerve syphilis, and only this. But there is a practical difference between syphilis of the nervous system in its active and inactive stages, and it is most important to discover with which we are dealing in a given case.

It is exceptional to find a well-marked case of advanced syphilis of the nervous system with normal pupils, and the Argyll-Robertson type is found so often as to require no comment. When it occurs alone, however, the sole sign and with no symptoms, then the physician is rightly puzzled as to its significance. I have examined 13 such cases where this was the sole sign, with no symptoms at all or symptoms which were only distracting to the diagnostician. Of these, 9 gave positive tests for syphilis, all except one strongly positive, usually both blood and fluid, so that the findings were unmistakable. Mr. J. E. C., a man of fifty, became irritable and could not concentrate on his work; he had a feeling of pressure through the temples, and was to all intents a neurasthene. His pupils were small, irregular, fixed to light, but reacted to accommodation. Tests were all

strongly positive. This in 1913; to-day, in spite of much intensive treatment, he is a patient at the McLean Hospital, a typical picture of dementia paralytica.

A physician at an institution was one morning accosted in this agreeable manner: "Why, doctor, your right pupil is dilated—you must have paresis." He consulted a neurologist, who found a typical Argyll-Robertson pupil and sent him to me for tests, which were strongly positive. Under thorough treatment with salvarsan tests have been rendered almost normal, and there is no evidence of advance of the syphilitic process.

Of four cases, however, which showed this type of pupil, tests were negative. What may we say of these? It is difficult to be dogmatic, but I believe they represent cases of arrested nerve syphilis, as seems quite certain in one, at least. On account of positive findings in the husband the wife asked for an examination, which revealed typical Argyll-Robertson pupils. On reviewing her story she remembered that, ten years ago, on consulting an oculist for diplopia, he had been much interested in her eyes, saying that they did not react properly; that at this time she had had some rheumatism, but that for the past ten years she had had absolutely no symptoms which might signify active tabes. Here, then, was almost certainly a case of tabes arrested in its early stages.

The same may be said, in a somewhat different sense, of 10 other cases in which Argyll-Robertson pupils were found in conjunction with other signs and symptoms of tabes, but in which all laboratory tests were normal. These cases, for the most part, suffer from pain, but fail to show advance of the disease, as shown by the cropping-out of new symptoms. They are probably stationary, so far as activity of the pathologic process is concerned, though degeneration of nerve-fibers probably goes on. These are, for the most part, tabetics of long standing.

What is the importance of tests in this group, if Argyll-Robertson pupils always signify syphilis of the nervous system, and of this view I have no reason to doubt. *It is in determining the stage of the disease*, whether it be an active, advancing process, or stationary and degenerative only, or dead and quiescent. In one case treatment may arrest the disease before it may fairly be said to have started; in the others there is no need of intensive antisyphilitic medication.

Irregular Pupils.—What can we say of irregular pupils? We have in our series cases with irregular pupils in whom tests are positive, and others in whom they are negative; but it is striking that, in the latter group, comprising 13 cases, in 7 there was either a history of lues or evidence of the disease. The negative tests, however, were useful in determining the importance of syphilis as an *active* factor in the symptom for which the patient presented himself, and in none have I reason as yet to feel that a mistake was made in saying syphilis is not the causal factor here. For example, G. S., a man aged forty-six, presented himself with a hemiplegia; he was euphoric, had a speech defect, his memory seemed poor, and his pupils were quite irregular. A diagnosis of early paresis was made. His tests were normal. This in 1913. If the diagnosis had been correct, he should now be well advanced and an unmistakable case; on the contrary, he is working, is much better than he was in every respect, and suffers only from the residual of his hemiplegia.

I was asked to examine A. M. because of bladder retention without evidence of local obstruction. The findings were, besides the retention, trabeculation of the bladder, irregular pupils, aortic roughening, and loss of sexual power, all very suggestive of syphilis. Both vascular and cerebrospinal tests were negative, and active tabes was excluded. Operation was undertaken in the hope of finding some local

cause which had escaped the examiners, and a peculiar, valve-like middle lobe of the prostate, impossible of detection by the finger or the cystoscope, was removed, with complete relief of retention.

So, with irregular pupils, we should, I believe, consider them as very often the result of syphilitic infection, but whether or not they are associated with *active* syphilis, or with syphilis *involving the nervous system*, should be left for the tests to decide.

NORMAL PUPILS—POSITIVE TESTS.

Twelve cases present absolutely normal pupils, so far as ordinary examination is concerned, and yet were the subjects of active syphilis, seven undoubtedly with involvement of the cerebrospinal axis. For the most part these were cases of recent infection, though not always.

While, then, we may regard irregular and unequal pupils as placing the patient under suspicion of syphilitic infection, it does not aid in telling us of the activity of the process. Conversely, a normal pupil does not preclude the existence of a severe syphilitic process, either of the vascular or cerebrospinal systems.

I will not take more time to analyze groups and subgroups. The above "eye cases," seen from the neurologic point of view, seem to me instructive. To those of us who have followed the diagnostic and therapeutic course of these patients the employment of lumbar puncture has become a more and more helpful—I might almost say an indispensable—procedure.

DISCUSSION.

DR. ARNOLD KNAPP, New York: We have had the opportunity, during the past year, of following a case of influenzal optic neuritis with the typical star figure in the macula, in which the examination of the lumbar fluid gave a cell count

of 21, with positive globulin. The Wassermann was negative in both the blood and the spinal fluid. The case was interesting as throwing light on the way in which the optic neuritis was produced. I should like to ask Dr. Ayer what significance he would place on these findings. After two weeks the cell count showed 14, with globulin zero; and the the last examination showed a cell count of 1. The case exhibited the usual course of the disease, and the patient gradually got well in ten weeks to three months.

DR. G. E. DE SCHWEINITZ, Philadelphia: While it is true that in most instances of exterior oculomotor palsy, if the patient does not recover within six months we are not likely to secure a favorable result, it is also true that in a certain number of cases, especially of syphilitic abducens palsy, a delayed response to active treatment may be followed, even after nine to twelve months, by complete recovery. I have observed this in two notable instances, and these and other cases form the bases of a recent paper by Dr. Spiller on this subject. These cases are interesting and novel.

It is important that Dr. Ayer has emphasized the danger of lumbar puncture in brain tumor—death has followed this procedure in more than one case.

DR. JAMES B. AYER, Boston, Mass.: In reply to Dr. Knapp, I would say that his patient might have had localized influenzal meningitis; on the other hand, he might have had a simple toxic condition. The lining of the spinal canal is a delicate affair, and it does not take much to cause a mild irritation of it, but not a definite meningitis. I have seen such in the course of typhoid fever. It was not a typhoid meningitis, but a toxic irritation in which, for a short time, there was an increase of proteids and an increased cell count. In that case the diagnosis depended on whether or not there were bacteria present, and also on whether the cells predominating were lymph-cells, showing an inflammatory process, or endothelial cells, showing a desquamative process.

TUBERCULOUS MENINGITIS WITH SPECIAL REFERENCE TO THE TUBERCLE OF THE CHOROID AND ITS PATHOLOGIC MANIFESTATIONS.

FREDERICK TOOKE, M.D.,
Montreal, Canada.

In considering the question of the miliary choroid tubercle, in spite of the recognition of an extensive expression of opinion on that small pathologic lesion, one is almost discouraged, at one's first investigations, by the varied findings of the several authorities. No less is one astonished at those who have overlooked a more complete reference to this important detail in the general treatise on tuberculous cerebrospinal meningitis. Bramwell, in Osler's "System of Medicine," 1910, volume vii, dismisses the question with the statement that choroid tubercles, when present, make the diagnosis certain. He states that Koplik found choroid tubercles in 9 of 46 cases in the first two days of illness. Bernstein found tubercle bacilli in the fluid in lumbar puncture in 100 cases out of 102 in which he looked for it. Batten, in Albutt's "System of Medicine," supplies one with a little more information, and says that ophthalmoscopically the two changes which require consideration in the disease are optic neuritis and choroid tubercles, to which, respectively, attention was first directed by Albutt and by Cohnheim. With regard to optic neuritis, Batten states that its indications in the early stage of the disease are often ambiguous, but late in the disease it can generally be detected on both sides, though unequally. Even then it is rarely severe or

comparable with that associated with cerebral tumor, but the increase of blurring of the edge and of curving of the vessels is conclusive to one who has observed the progressive change in a given case.

In judging of the progress of the neuritis, Garlick lays great stress in these cases on the alterations which take place in the apparent lumen of the arteries and veins, and on their altered relation to one another. Batten further states that tubercles of the choroid can be observed occasionally in successive development at the various parts of the fundus, beginning as minute round dots which gradually become opaque in their centers and sometimes confluent at their margins. Tubercles of the choroid that seem to develop during the course of a brain illness are so far in favor of that illness being tuberculous meningitis. He found that, out of 16 consecutive cases of choroid tubercles, 13 showed post-mortem evidence of tuberculous meningitis, while 3 presented tubercles in other organs, but no tubercle in the pia mater. Thus, apart from any other sign, tubercles of the choroid cannot be taken as indicating that there is necessarily a deposit also in the pia mater, but it bears witness probably to a wide-spread tuberculosis.

Marple, in an article read before the Congress of Ophthalmology at Oxford in 1912, made the assertion that tubercles are to be seen in every case of tuberculous meningitis. In Gower's work on "Medical Ophthalmoscopy," in which one must at once associate the collaboration of the late Marcus Gunn, the statement is made that tubercles of the choroid may now and then be found in tuberculous meningitis. Cohnheim would have us believe that these tuberculous bodies are less frequent in tuberculous meningitis than in generalized tuberculosis without meningitis. In contradiction to Marple's findings Heinzel, after examining 41 cases of tuberculous meningitis, was unable to convince himself that tubercles were present in a solitary instance, while

Garlick, after repeated examination of 26 cases, was satisfied with their presence in only a solitary instance.

Again, Marple's figures are supported in part by the findings of Carpenter and Sydney Stephenson, who are satisfied that they are present in 50 per cent. of the cases, while further evidence is attached by the figures of Bock, who places the percentage at 82.7 and Leiter at 75.

There is no doubt in the mind of the writer that much facility in examining the fundi of these cases has been afforded by the various patterns of electric ophthalmoscope, allowing at once more frequent examinations with a minimum amount of effort, as well as bringing out more brilliantly illuminated details of the interior of the eye. Yet, on the other hand, one cannot but feel that such brilliancy of illumination, even with a frosted globe, produces strong light reflexes thrown by the filaments of an electric lamp as well as those cast by the walls of the vessels on the retina, and which produce impressions on the mind of the observer. These impressions of such minute lesions one is apt to interpret as pathologic lesions. In my examinations I have consequently tried to avoid such possible fallacies where opportunities were favorable, and have used the electric ophthalmoscope in conjunction with the less brilliant but possibly less disguised picture of a reflected image with a Morton instrument.

In the early stage of the disease, where much restlessness and opposition to examination are displayed by the patient, and where an examination of the fundi is rendered more or less difficult, the electric ophthalmoscope is a manifest advantage. In my experience, however, as my figures will subsequently show, findings of importance are generally not noted during the period of incubation, but rather toward the end of the disease. In spite of convulsive spasms which may ensue shortly before exitus, much less actual resistance is met with. The method generally employed is to place a

clean towel or a piece of gauze over the lower half of the patient's face, in order to render a rather unpleasant examination as hygienic as possible when adopting the direct method. In cases toward the end of the disease, where exfoliation of the corneal epithelium has begun, I have a saline solution applied to the eye by an assistant from a small drop-bottle. I have used this method also very shortly after death, before colloid changes have occurred in the vitreous, in an endeavor to define possible tubercle formation which I could not satisfy myself existed up to the time of death. Although it is frequently unnecessary, a drop of atropin is instilled into each eye every couple of days in order to facilitate the examination.

I have considered myself fortunate that my efforts in an investigation on this subject have been able to be carried out in the wards of a large general hospital. Many of the authorities whom I have quoted have based their statistics on examinations made in children's hospitals. Although a recognized eye hospital is a desideratum in every community, it cannot be denied that a properly equipped and conducted eye clinic in a general hospital will frequently afford opportunities for special study of the fundus oculi requisitioned from other services. It may consequently be interesting to know that, from the figures which I have collected from the records of the Royal Victoria Hospital, Montreal, tuberculous meningitis is far from being confined to childhood. It is quite true that young children form a fairly large proportion of all cases, but the disease, as generalized tuberculosis itself, may be present at any age. I have seen cases of tuberculous meningitis in the fifth and sixth decade where rather obscure conditions clinically were finally cleared up postmortem, and in one case, at least, where definite clinical diagnosis was established only by the detection of choroid tubercles with the ophthalmoscope.

In 102 cases which I have collected there were 60 male and

42 female patients. The ages of these patients may be classified as follows:

Between	1	and	2	years	7
"	2	"	5	"	23
"	5	"	10	"	15
"	10	"	15	"	12
"	15	"	20	"	14
"	20	"	30	"	15
"	30	"	40	"	9
"	40	"	50	"	2
"	50	"	60	"	5

It is a matter of regret that in the 102 cases which I have collected only 73 were referred from the Department of Internal Medicine to the section of Ophthalmology. Regarding the condition of the fundus oculi, the findings are, in consequence, less complete than had a routine examination been made by a competent authority in each individual case. A few cases are recorded both of negative and of positive findings by medical house officers, but most of us who have served in such a capacity in our early experience know and readily admit that our skill and reliability are hardly commensurate with our good intentions in an examination of this nature at this particular period.

Of the 73 cases examined by members of the attending staff, choroid tubercles were present in seven cases. In every instance they appeared not earlier than three days before death. In one case repeatedly and separately examined by each member of the attending staff the mere suggestion of a solitary tubercle formation was first noted by the writer three hours before death. This condition was verified post-mortem, and I am privileged in being able to demonstrate sections of the eye from this patient (Fig. 1).

Only too frequently, however, cases have been examined but once or twice at what one might consider a considerable interval of time before exitus, with negative findings regarding the presence of tubercles. It is further to be regretted that many of these cases were not followed more frequently.

In a number, however, which I have been privileged to study, I have followed them up to within three or four hours prior to death, feeling that tubercles might appear as a final manifestation. The two cases which I examined last month,—one six hours and the other three hours before death,—in each case there was present a slight degree of perineural edema, as I have generally noted in cases with an actual tuberculous formation, but in these two cases up to that time tubercles were not present. In these cases, as in others examined late in the course of the disease, and where I had felt a certain degree of misgiving and discouragement regarding my ophthalmoscopic findings, I followed the case to autopsy, only to have my clinical report substantiated.

It may be held that these tubercles have a peculiar faculty of evanescence, manifesting themselves clinically, only to disappear prior to death. Although allowing that fresh tubercles may possibly make their appearance, and perhaps even that some may vary in size and position, it has been my pathologic experience that cases which I have seen and the sections which I am showing in this contribution correspond exactly postmortem to the ophthalmoscopic findings.

Early inflammatory changes about the nerve, described by some as a perineuritis, by others rather as a perineural edema, were recorded in 30 cases of 73 examined. This manifestation was reported varying from five days to one day before death, and when present, the tubercles of the choroid were invariably an antecedent to the latter condition. Changes of this nature I have generally found to be associated with conditions of a basal type of meningitis, where the disease progressed forward to the optic chiasm, frequently following the sheath of the cranial portion of the optic nerve up to the sphenoid fissure (Fig. 2).

Regarding the presence of choroid tubercles, could our experience be that of Marple, or even that of Carpenter and Stephenson, it might be held as a strong point in the diagnosis

of many obscure cases. That they have been known to be absent in many cases postmortem, with active tuberculous lesions in the meninges, would offset their diagnostic value as a negative factor. In adult cases their presence may shed light on what has been regarded as an obscure condition.

PATHOLOGY.

Regarding heredity as an etiologic factor, it is absolutely useless to discuss. In a number of cases it is true that a clear and definite family history of tuberculosis could be elicited, and in many instances was candidly admitted. In many cases of foreigners, particularly those of Jewish extraction, who, by the way, constituted 21 per cent. of the total number, where very intelligent information could not be obtained relative to their prior health and to that of their progenitors, it was absolutely impossible to have any of them admit of being victims of any of the ills that flesh is heir to. This may be attributed to ignorance or to the unnatural caution and suspicion of such a class.

It is not the intention of the writer to attempt to present anything resembling a complete treatise on the general pathologic lesions found in tuberculous meningitis. Such a course would be far from the original idea of this paper. Incomplete postmortem examinations were made in 48 cases, and of these, 38 included the head, while in 5 cases permission was refused that most important part of the anatomy for study and observation. I may say at the onset that there was no case of tuberculous cerebrospinal meningitis without other manifestations of tuberculosis elsewhere in the body. There was, further, no such case as a primary miliary tuberculosis of the meninges.

To enumerate the foci of coincidental lesions would be to include the whole human organism. As is the case of tuberculous lesions in general, so in this particular manifestation of a general disease, evidences were most frequently found in

Fig. 1.—Female patient, aged eighteen. First manifestation appeared twenty-four hours before death as a tiny, pin-head point of hyperemia, less diffuse at its periphery. Definite tiny, pearl-gray body replaced it three hours prior to death. Section shows gradual occlusion of choroid blood-vessel, with circumscribed infiltration of lymphocytes and an intervening area of fibroblasts which appear to be approaching the stage of caseation.

Fig. 2.—Same patient as Fig. 1. Section of intracranial portion of optic nerve near sphenoid fissure. Clinically a slight degree of perineural blurring was noted. Sheath of optic nerve studded with innumerable miliary tubercles, which at one point had coalesced to form a tuberculous granuloma. Separate layers of optic nerve-sheath included in one granulomatous mass. Dense infiltration of leukocytes between sheath and optic nerve itself, with infiltration along trabeculæ of optic nerve. No giant-cell formation.

the lungs; in fact, in no isolated case where a general postmortem examination was performed, not even in early childhood, was the chest cavity found to be free from tuberculous disease. One case in particular, suggesting the hematogenous nature of the infection, showed an old tuberculous focus in the lung which had relighted and had broken directly through the wall of one of the pulmonary vessels. It has been rather interesting to note coexisting manifestations of ulceration in the large or small bowel in 17 per cent. of the cases.

A description of the usual pathologic findings of a case of meningitis due to tuberculous diathesis is, as I have already said, made unnecessary. But I will confine my remarks to a few of the commoner lesions which were recorded about the base of the brain. Changes of a pathologic nature directly involve the base of the brain in so far as one could determine, microscopically speaking, in all but two cases. In these two exceptions no fundal changes were noted. As a rule, the brain was inclosed in its meningeal sheath in a turbid, greenish, grayish fluid, which frequently assumed a fibrogelatinous appearance at the base of the organ. This fluid differed distinctly in its postmortem appearance from that obtained from lumbar puncture prior to death, where it, as a rule, appeared perfectly clear.

Miliary tubercles were noted in varying numbers, sometimes very few, but when present, they were localized toward the base. When the structures at the base of the brain were involved, it is interesting to notice the number of miliary tubercles which seemed to have a predilection for the fissure of Sylvius. Tubercles at the base varied in size, also in position, from the sheath of the cranial portion of the optic nerve forward to the cerebellum behind. In one case a tuberculoma of the right optic nerve was present, and the eye exhibited a well-defined perineuritis, as well as a small solitary tubercle of the choroid, after very late clinical

manifestations (Figs. 1 and 2). Two cases of coincident tuberculoma of the cerebellum with basal manifestations of miliary tuberculosis had not shown any unusual signs in the fundi prior to death. In those cases which were sufficiently extensive as to include the vessels at the base of the brain it was interesting to note how frequently these were studded along their course by these small miliary bodies. Direct reference has been made to them in almost half of the cases, and with fundal changes occurring in many of them it must suggest a hematogenous source of infection in the case of the tubercles at least. Their appearance, however, does not mean that perivascular tubercles had of necessity to be responsible for the presence of changes in the fundi, for it is worthy of note that, in a number of instances where such post-mortem changes were remarked upon, negative reports had been made of the fundus only a short time before death occurred. It consequently seems that these changes in the fundi are, in all likelihood to a very large extent, due to a hematogenous infection. At the same time it is quite impossible to exclude sources of infection by way of the perivascular lymph-spaces, as certain of my sections would seem to point out. Von Graefe has stated that the optic neuritis found in tuberculous meningitis is a descending one, and due to a direct extension from the pia-arachnoid space through the trabeculæ of the optic nerve, setting up direct inflammatory changes. In the 30 cases manifesting changes about the optic nerve there was only one on record which showed anything at all resembling a high degree of inflammation, the changes being rather those of venous engorgement with perineural blurring about the margins of the disc. I have never seen in any of these cases a choked disc of measurable swelling. My explanation for such clinical findings is that the changes about the optic nerve are not altogether through the trabeculæ of the optic nerve and along the course of the nerve-fibers, as certain authorities would have us

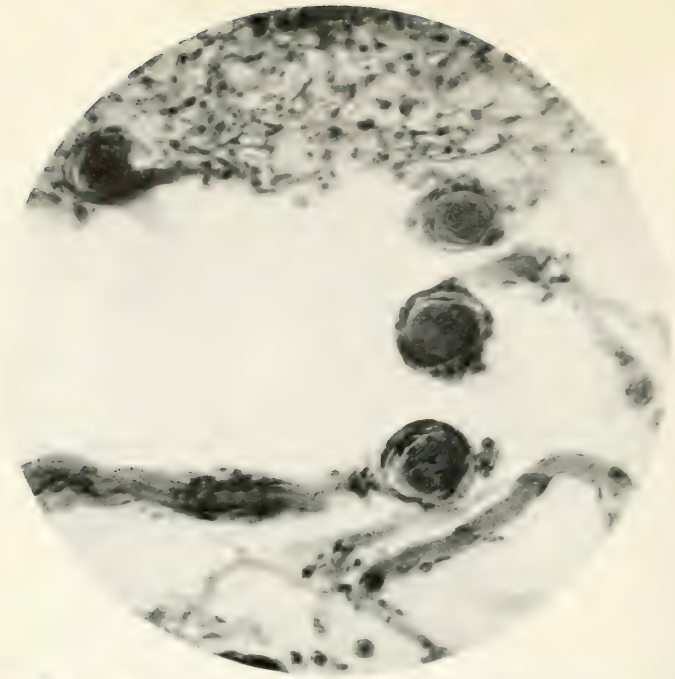


Fig. 3.—Section from same patient as Fig. 2. Orbital portion of optic nerve close to the eyeball. Clinically a slight condition of perineural blurring was noted. Slight infiltration of pia-arachnoid coat of nerve-sheath, with very moderate infiltration of leukocytes from it into vaginal space, as contrasted with cranial portion of nerve. Tiny blood-vessels distinctly engorged with transudate of lymphocytes through vessel-wall, the nuclei of perivascular tubercles.

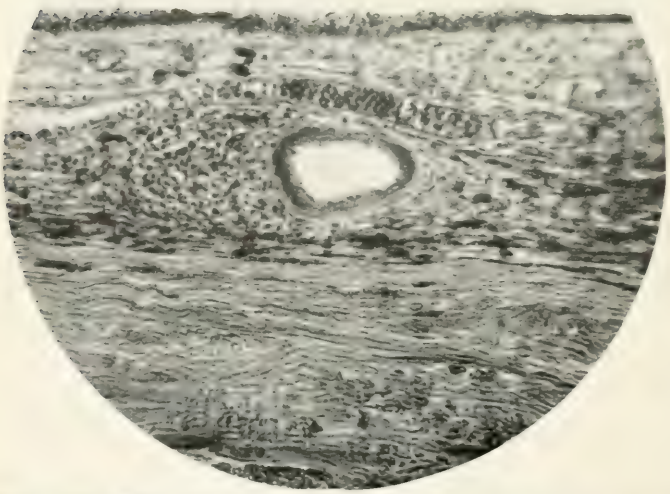


Fig. 4.—An early pathologic manifestation of a tubercle of the choroid. Engorged choroid capillary, with exudate of leukocytes through vessel-wall. Dilated blood-vessel, which is showing a condition of perivasculitis of a fairly advanced degree.

believe. In the cases to which I have already referred, where a tuberculoma existed about the cranial portion of the sheath of the right optic nerve, the condition was further involved by the presence of innumerable tubercles. Sections through this portion of the nerve show an absolute caseation involving all sheaths of the nerve, with an intense infiltration completely occupying the space between the nerve and its sheath, as well as following its ependyma. This condition was confined to that portion within the cranial cavity over which the tubercles are situated (Fig. 2). That portion without the cranial cavity and within the orbit shows no such similar microscopic appearance, this seemingly having been arrested in the neighborhood of the sphenoid fissure. There is consequently no such condition as direct filtration through the vaginal space. The orbital portion of the optic nerve shows there is direct infiltration of the pia-arachnoid, but nothing more. Such an intense condition as that found within the cranial cavity is nowhere noted, and nothing at all resembling caseation occurs. In fact, tubercle formation is not present. The tiny vessels of this portion of the optic nerve sheath, however, are frequently engorged, and one can distinctly, in a number of instances, make out in the vaginal space tiny capillaries showing an egress of lymphocytes through their walls to be possibly thrown free into this space or to constitute the nucleus of subsequent perivascular tubercle formation (Fig. 3).

Regarding the structures of the choroid tubercle itself, I have found that they vary somewhat in size from 0.5 to 2 mm., the smaller ones generally being the more common. They are generally found, in my experience, in the posterior hemisphere, and, as a rule, near the bifurcation of one of the larger retinal vessels. The first manifestation of a tubercle involving the choroid is that of a tubercle elsewhere. Engorgement of a vein or of a small capillary through which a subsequent migration of lymphocytes occurs, from this

procedure there is constituted a condition of perivasculitis, which in due time encircles the whole lumen of the vessel (Figs. 4 and 5). Following this perivascular infiltration of lymphocytes changes occur in the blood-vessel itself, which may vary as follows: In the first place, it may merely be strangulated by the pressure of its encircling zone for inflammatory change, at the same time cutting off all nutrient supply to the vessel itself; in the second, a hyperplasia of the media may have a great deal to do with excluding its lumen; and, finally, an endarteritis obliterans may ultimately close up this whole focus from subsequent blood-supply.

With the infiltration of lymphocytes there occurs a coincident infiltration of fibroblasts, and, no doubt, should the lumen of the vessel become completely occluded, caseation would in due course ensue. Although some of my sections show such vascular processes well on toward completion, yet in no instance was I able to determine that they were such as to show such actual microscopic changes, although I believe that such changes have been noted by other observers. This condition would naturally be noted in cases where tubercles had made an early clinical manifestation, rather than in those which I am showing where they appeared later on in the disease.

Giant-cell formation is typically shown in two cases without there being of necessity a process of caseation. Tubercle bacilli have been noted in several instances, but, being of a rather frugal mind, I have preferred to preserve my rather scanty material for the preparation of sections and to accept the statement and manifestations of the sections provided by my friend, Dr. Brown Pusey, to whom I wish to record my sense of appreciation. It is interesting to note, in regard to the formation of miliary tubercles of the choroid, how closely they are confined to the vascular elements of the eye. In every instance which I have so far seen the infiltration

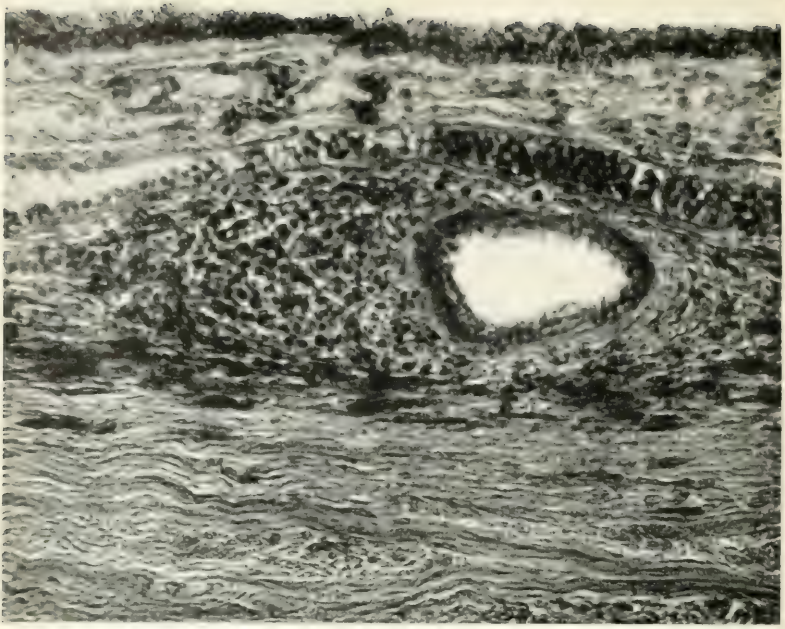


Fig. 5.—Same as Fig. 4, only higher magnification. Section clearly shows egress of lymphocytes through the capillary wall, forming the zone of perivasculitis or infiltration about a dilated vein.

Fig. 6.—Male patient, aged seventeen. Tubercle first noted clinically twenty-four hours before death. Tubercle confined entirely to choroid coat, and consists of a peripheral zone of infiltration in which are included fibroblasts, through which are scattered numerous giant-cells. Caseation has not occurred to any appreciable degree. The pathologic lesion is confined entirely within the membrane of Bruch, and does not extend backward from the choroid to the sclerotic coat.

never seems to have gone beyond the region of the choroidal blood-vessels, the pathologic process being invariably included within the delicate hyaloid membrane of Bruch, on the one hand, and within the scleral coat, on the other, and I have never seen the slightest evidence of infiltration between the membrane of Bruch and the retina (Fig. 6).

Regarding the pathology of cerebrospinal fluid, this, in my opinion, may be accepted as a much more reliable source of information for the establishment of a clinical diagnosis. In all cases a definite leukocytosis was present, the lymphocytes being invariably in the ascendant. One case was, however, rather misleading, polymorphonuclears being more numerous than the lymphocytes, in the proportion of 70 to 30 per cent. In another case they were equally divided, but both cases show definite pathologic lesions at the base of the brain, as well as elsewhere in the body, of a distinctly tuberculous nature. In the vast majority of cases, however, the proportion of lymphocytes was well over 75 per cent., and in more than one case rose as high as 100 per cent. The degree of lymphocytosis would not appear to have any direct relationship to coexisting manifestations in the choroid or the optic nerve.

REFERENCES.

Bramwell: Osler's System of Medicine, 1910, vol. vii.
Batten: Clifford Albutt's System of Medicine, 1910, vol. viii.
Marple: *The Ophthalmoscope*, October, 1912, p. 559.
Gowers: *Medical Ophthalmoscopy*, 1904, p. 164.
Heinzel: *Jahrbuch f. Kinderheilkunde*, 1875, p. 334.
Garlick: *Medico-Chirurgical Trans.*, 1879, p. 441.
Manz: *Arch. f. Ophthalmologie*, 1858, vol. iv, 2; 1863, vol. ix, 3. *Klin. Monatsbl. f. Aug.*, 1881, vol. xix.
Bock: *Virchow's Archiv*, xci.
Cohnheim: *Virchow's Archiv*, xxxix.
von Graefe: *Arch. f. Ophthalmologie*, ii, 1855.
Parsons: The Pathology of the Eye, 1905, vol. ii, p. 462.
Greeff: Orth's pathologische Anatomie, 1903, part x, p. 312.

DISCUSSION.

DR. W. B. MARPLE, New York: I made a contribution to this subject before the Oxford Ophthalmological Congress

in 1912, which was published in the *Ophthalmoscope* for October of that year. I might say that all the cases that I have seen were babies; that is, children under two years of age.

Previous to 1912 I had never been able to discover choroidal tubercles in more than 4 per cent. or 5 per cent. of the babies (admitted to The Babies' Hospital) suffering from tuberculous cerebrospinal meningitis. I then determined to make the examinations of these patients more frequently, and see if I could verify the statistics of Carpenter and Stephenson (50 per cent.). This was rendered possible by having my own examinations supplemented by others made by the resident house physician, who soon became adept in the use of the electric ophthalmoscope, and also very much interested in the subject. From then on a series of 13 cases were examined, and in *every* one choroidal tubercle was discovered. In several cases on successive days one or more tubercles were found which were not present the day before. One case I examined carefully at 6 P. M. and found nothing; at 10 P. M., four hours later, the house physician telephoned me that he had just discovered a tubercle where nothing was present four hours previously, and his observation was corroborated by myself the following day.

In other forms of choroiditis, such as exudative, we generally find a more or less numerous exudate on Descemet's membrane. Although I have looked for this repeatedly in these cases of tuberculous meningitis with tubercles of the choroid, I have never found it in a single case. A mild papillitis has been observed in a little less than half of the cases.

The tubercles have been generally located in the neighborhood of the optic disc (within one to three disc diameters from the latter). In the greater number of cases they are found in one eye only—often we find one, less frequently several. Most of those I have seen have been one-fourth the diameter of the disc or smaller. They have been small miliary tubercles, round or oval in shape, of a grayish color, usually without any pigment, as though the retinal pigment epithelium had been gently brushed away. Retinal vessels are frequently seen passing undisturbed in outline across their surface. The cases are usually under observation

only a short time, and not infrequently are moribund on admission.

My conclusions as to the frequency with which choroidal tubercles are found in these cases have been confirmed by the further observations of the last three years. As Mr. Stephenson has said, it all depends on your house physician. If he is careful and painstaking, and examines the cases often enough and the cases live long enough, you will find choroidal tubercles in practically all. If he is careless or indifferent, or the cases die shortly after admission, you will find the condition infrequently.

DR. R. L. RANDOLPH, Baltimore, Md.: It is evident that frequent examinations are necessary to convince one that a tuberculous condition exists in the eye-grounds. I cannot say how many cases I have examined in the Lane Children's Hospital in Baltimore. For a long time I was faithful in making repeated examinations, but failed to find the changes described by Carpenter, Marple, and others. I have been constant in examining the children in the last stage of life. We know that the appearance of these tubercles in the choroid is a terminal event in the history of the meningitis. Since January, however, I have come across two cases. They were so gross, so clean cut, so unmistakable, that one could not regard them as probably anything else but tubercles. Both children were under one year of age, and both died. These were the only two cases of tuberculous choroiditis which I have seen. I should like very much to hear from others—those who have been associated with large hospitals—as to the impression they have obtained from their experience along these lines. I was very skeptical for a long time myself. I tried to substantiate the findings of the men who have written upon the subject, but was very much disappointed at not being able to find the tubercles.

I should like to ask Dr. Marple whether he has confirmed any of his cases by histologic examination.

DR. MARPLE: There was published the result of the histologic examination in one patient. It was a rather unusual case, with a large tubercle near the disc, and section showed the giant-cells.

Dr. Carl Koller, New York: I have had for many years the opportunity of examining children in the children's ward of Mt. Sinai Hospital, and have found that those with tuberculous meningitis show, as a rule, tubercles of the choroid at some time toward the fatal end of the disease. My experience was similar to that of Dr. Marple. The more frequently and thoroughly the cases were examined, the more it became apparent that it is rather an exception if a child near death from tuberculous meningitis does not show tubercles in the choroid.

Dr. George S. Derby, Boston, Mass.: The beautiful preparation which Dr. Tooke gave me a chance to examine explains to me, for the first time, those cases of proliferating retinitis and perivasculitis of tuberculous origin occurring in young adults, and shows very clearly how hemorrhages and other such processes take place in the eyes in such cases.

MALIGNANT UVEITIS TREATED WITH THYROID EXTRACT.

JAMES BORDLEY, JR., M.D.,
Baltimore, Md.

There are several well-recognized varieties of uveitis, ranging from the mild form, seen as an evidence of senility, to the malignant choriocyclitis. As the cases which are to be considered here belong to a rather rare form, I shall speak of their symptomatology.

Except for intensity, the symptoms were the same in all of the patients. The course of the disease was slowly progressive. The first evidence of trouble was photophobia; this was followed by loss of visual acuity. Coincident with the failure in vision exudates appeared on the posterior corneal surface. These exudates were of two forms: small dots located near the inferior border, in places becoming confluent and forming irregular masses; and large plaques with a

Fig. 1.—Malignant uveitis, showing conjunctival cysts and corneal deposits.

dense, opaque center and translucent edges. The latter seldom exceeded four or five in number, and frequently disappeared, leaving scar tissue to mark their former location (Fig. 1).

Dilatation of the pupil and a greenish discoloration of the base of the iris were early symptoms. The discoloration of the base we regarded as an atrophy, as the iris apparently never regained contractility and reacted neither to light nor to accommodation. When the conjunctiva was closely studied by oblique illumination and magnification, a large number of cysts were found in the bulbar portion, encroaching in places on the epithelial layer of the cornea.

The formation of the vitreous cloud in the cases watched from the start of the disease was very curious. It looked at first not unlike a thin, dotted veil suspended in the vitreous chamber. The mesh of the veil became more and more coarse, and the dots larger and larger until the whole formed a homogeneous, translucent mass, filling the chamber and obscuring the retina.

In one case the optic disc was slightly hazy, and in all five engorgement of the retinal veins was noted. The intraocular tension, at first normal (20 to 25), increased as the disease advanced, in one case reaching 45. There was in every instance a secondary decline, dropping in two cases to five.

All the patients complained bitterly of photophobia; the eyes were tender to touch, and there was limited injection of vessels of the deep circumcorneal zone. The fields of vision were normal until dimmed by vitreous haze.

A careful physical examination was carried out in all these patients by eminently qualified internists, and in not one was a lesion found in any of the so-called vital organs. Wassermann tests were made in every case by Dr. Albert Keidel, who is in charge of the department of syphilis at the Johns Hopkins Hospital. The results of his work will be taken up in the case reports.

In every case there was a positive tuberculin reaction—the von Pirquet and injections of "old" tuberculin were both used. In three of the patients the reaction to "old" tuberculin was very marked, both constitutionally and in the diseased eye. In all the percentage of hemoglobin and the red-cell count indicated anemia. The kidney functions were normal, and in but one case did we discover albumin. The mouth, nose, and throat of every case were carefully studied, and *x*-ray pictures made of the teeth and the nasal sinuses.

The treatment of the ocular condition of these patients included: mercury, iodid of potassium, pilocarpin, and sweat-baths. As a positive tuberculin reaction, a low-grade afternoon temperature and loss of weight had to be considered in each instance; fresh air, milk, eggs, and rest were insisted upon. Subconjunctival injections of sodium chlorid and bichlorid were given without the slightest apparent improvement except for the relief of pain.

There was one medicine used the results of which so impressed all the physicians associated in the treatment of these patients that I feel justified in calling attention to it. I speak of thyroid extract. A fairly large and by no means pleasant experience in the treatment of malignant uveitis has convinced me that, in these cases, we have to deal with two factors: an infection and an eye that makes a strong defensive, but lacks the something necessary to an offensive, fight. Wishing to test this hypothesis, I started, several years ago, to feed patients suffering from uveitis with extracts of various tissues of the eyes of sheep, hogs, and calves. The sum of these experiments was failure to accomplish anything. Still having faith I fed patients on the chemicals necessary for tissue up-building, and with no more success. It was about this time that I was fortunate enough to see a patient, who, with malignant uveitis and a positive Wassermann, resisted the most strenuous antisyphilitic treatment.

This patient also had patches of thickened skin, was morose, and suggested the possibility of a rather atypical myxedema. Thyroid extract was ordered, with a hope that it might be of help in the treatment. The change for the better was so immediate and striking that it prompted me to make a search for patients with outspoken hypothyroidism. I had the privilege of examining a fairly large number of such cases, and found two with active uveitis and four with lesions clearly the result of former malignant uveitis. I also ran across the history of another case in which, following a thyroidectomy, "extensive vitreous changes" made their appearance. This latter case interested me because, with the introduction of thyroid extract, made necessary by other symptoms, the vitreous opacities disappeared. That my observations were not the first to point to the relationship of hypothyroidism and uveitis is made quite clear by an article on the subject by Demets.

I do not wish it inferred, from what I have said, that I believe that malignant uveitis is necessarily a symptom of cretinism or myxedema. My own experience has left quite a different impression; indeed, I have seen one case, a patient of Dr. Howard A. Kelly, on whom a panhysterectomy had been performed, resist all thyroid treatment and clear up only after taking ovarian extract for about two months; and another patient who responded neither to thyroid nor to any other treatment. My only idea in presenting this subject is to show the successful results of the use of thyroid extract in patients suffering from the type of malignant uveitis which I have outlined above.

In all, eight patients have been treated, four with marked success, two must be classed as doubtful, and two as failures. Of this number, I am able to discuss only five, as the records in the other cases are not yet completed.

The charts illustrate the effects on vision—and the vision was a good index of the progress of the disease.

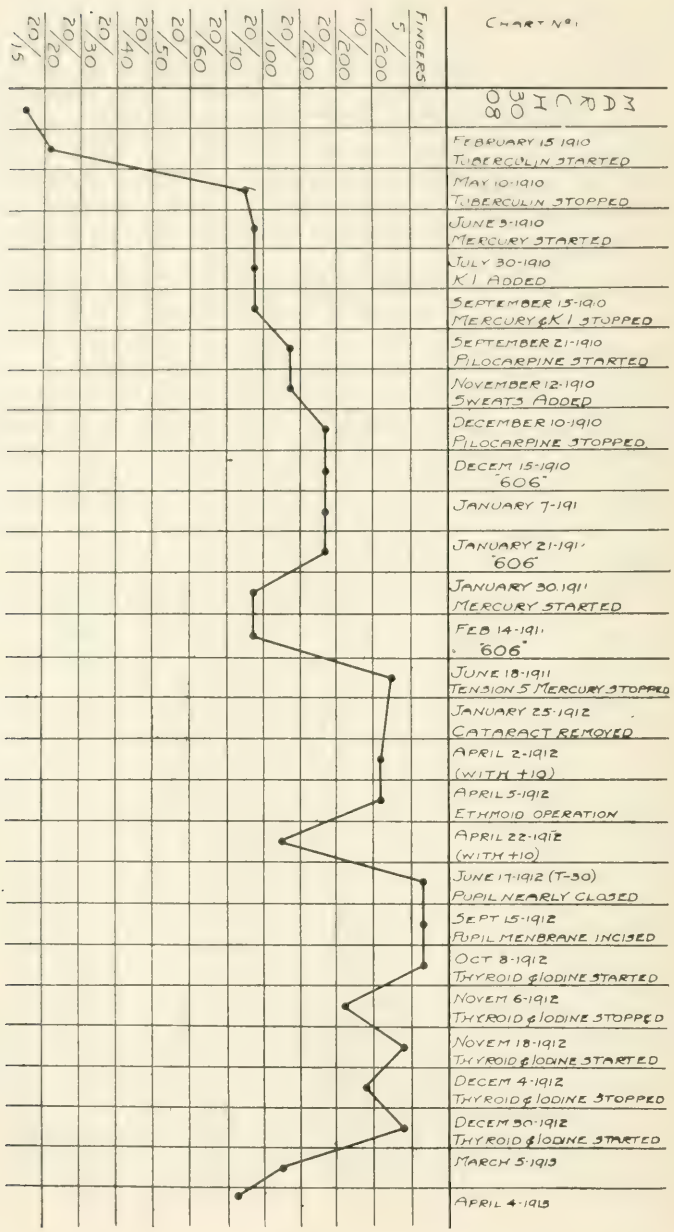

20/15
20/20
20/30
20/40
20/50
20/60
20/70
20/100
20/200
10/200
5/200
FINGERS
CHART No. 1
OD
OS
FEBRUARY 15 1910
TUBERCULIN STARTED
MAY 10-1910
TUBERCULIN STOPPED
JUNE 3-1910
MERCURY STARTED
JULY 30-1910
K I ADDED
SEPTEMBER 15-1910
MERCURY & K I STOPPED
SEPTEMBER 21-1910
PILOCARPINE STARTED
NOVEMBER 12-1910
SWEATS ADDED
DECEMBER 10-1910
PILOCARPINE STOPPED
DECEM 15-1910
"606"
JANUARY 7-191
JANUARY 21-191
"606"
JANUARY 30 191
MERCURY STARTED
FEB 14-191
"606"
JUNE 18-1911
TENSION S MERCURY STOPPED
JANUARY 25-1912
CATARACT REMOVED
APRIL 2-1912
(WITH +10)
APRIL 5-1912
ETHMOID OPERATION
APRIL 22-1912
(WITH +10)
JUNE 17-1912 (T-30)
PUPIL NEARLY CLOSED
SEPT 15-1912
PUPIL MEMBRANE INCISED
OCT 8-1912
THYROID & IODINE STARTED
NOVEM 6-1912
THYROID & IODINE STOPPED
NOVEM 18-1912
THYROID & IODINE STARTED
DECEM 4-1912
THYROID & IODINE STOPPED
DECEM 30-1912
THYROID & IODINE STARTED
MARCH 5-1913
APRIL 4-1913

Chart 1.

CASE I.—Woman, aged forty-five years, who, in spite of treatment, lost both eyes. The left eye was first affected, and in 1907 she presented herself with a history of decline in vision in that eye, following a severe attack of tonsillitis and rheumatic fever in 1906. She had all the symptoms outlined above in an intense form; the vision was reduced to counting fingers, and the tension was noted as "high normal." A physical examination made by Dr. T. R. Brown was negative, except for some bad teeth and tonsils. Wassermann reaction negative and tuberculin reaction positive. The teeth and tonsils were removed, and the latter were examined for evidences of tuberculosis, but without success. Injections of salicylate of mercury were given, and the patient sent to the mountains. The disease progressed, and the patient was brought home, given pilocarpin and sweat-baths, without obtaining any benefit. The tension of the eye gradually declined, the pupil became closed by an exudative mass, and in spite of operations and medicines the eye was lost.

After a lapse of three years the right eye became affected and developed the same malignant type of uveitis as the left. As the treatment of the right eye had utterly failed, it was determined to adopt a different line of procedure with the left, so small doses of tuberculin were given for three months, most of which time the patient was kept in bed and gorged with milk and eggs. During this period the eye steadily lost ground, both objectively and subjectively, so it was thought unwise to continue the tuberculin. Courses of mercury, pilocarpin, and later salvarsan were instituted. No improvement was noted except from the last, and this was only transitory. During the first months the tension was "high normal," then a steady decline was noted, the tension falling as low as five.

After being under constant observation for fourteen months she drifted away and sought the consolation and assistance of "Christian Science." Some months later she again came for consultation. It would be hardly fair to say that the fully mature cataract which she then had resulted from the efforts of the "healer," but whether this was so or not it necessitated an operation, which, as was to be expected, helped but little.

In October, following the cataract operation in January, thyroid extract in one-grain doses was given twice a day. The vision improved sufficiently in one month to enable her to come to my office alone. Unfortunately, in spite of the gain in vision, the thyroid had to be from time to time discontinued on account of loss in weight and strength, and eventually had to be given up, as a point was reached where fainting attacks and palpitation became alarming. At the time the extract was stopped the vision had improved from counting fingers to $\frac{3}{7}\frac{0}{0}$, and it was indeed discouraging, to say the least, to return to mercury and watch the eye become blind.

Case II.—Girl, aged nineteen years, noticed a decline in vision starting about June, 1912. She was first seen October 8th. She then had a double chronic ethmoiditis, a positive Wassermann, and a positive tuberculin reaction. The conjunctiva of the left eye was somewhat congested and filled with small cysts; many dots were to be seen on Descemet's membrane; the base of the iris was greenish in appearance, the pupil semidilated and did not respond to light, and the vitreous was a dense black mass.

As a physical examination made by Dr. T. R. Boggs and a gynecologic one by Dr. Guy L. Hunner gave no additional help, the patient was given mercury and salvarsan, but without benefit, for two months. Pilocarpin and sweat-baths were used for two months without hindering the progress of the disease or easing the pain. She was then given small doses of thyroid extract and iodin, and, as they were well borne, the dose was increased to four grains of thyroid and one grain of iodin per day. After reaching the maximum dose it was necessary, now and then, to discontinue this treatment for short periods. Its effect on the progress of the disease can briefly be described by saying that her vision from February to September increased from $\frac{5}{2\,0\,0}$ to $\frac{2}{7}\frac{0}{0}$; the vitreous cleared, and all inflammatory signs disappeared, leaving only a small corneal opacity, the result of organization of one of the large exudative masses on the posterior surface of the cornea.

On June 21, 1913, her ethmoids were opened and polypoid

tissue removed. This did not, so far as could be told, materially influence either the eye changes or the vision.

Practically two years have elapsed since the thyroid was

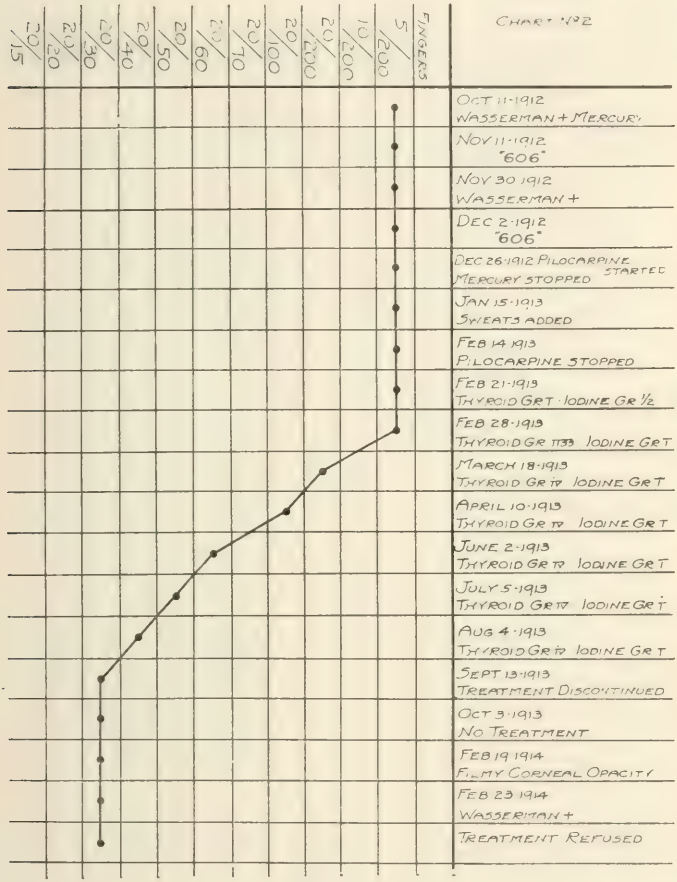


Chart 2.

discontinued, and in spite of a positive Wassermann she has had no return of her trouble. Unlike the first patient, she steadily improved in health and weight while taking the thyroid.

CASE III.—Nurse in the Johns Hopkins Hospital, following an acute attack of tonsillitis and rheumatic fever, developed within one month intense photophobia and a "red eye." She was in Canada on her vacation and did not report for examination until the vision of the offending eye, the left, had been much impaired.

A physical examination was made by Dr. W. S. Thayer,

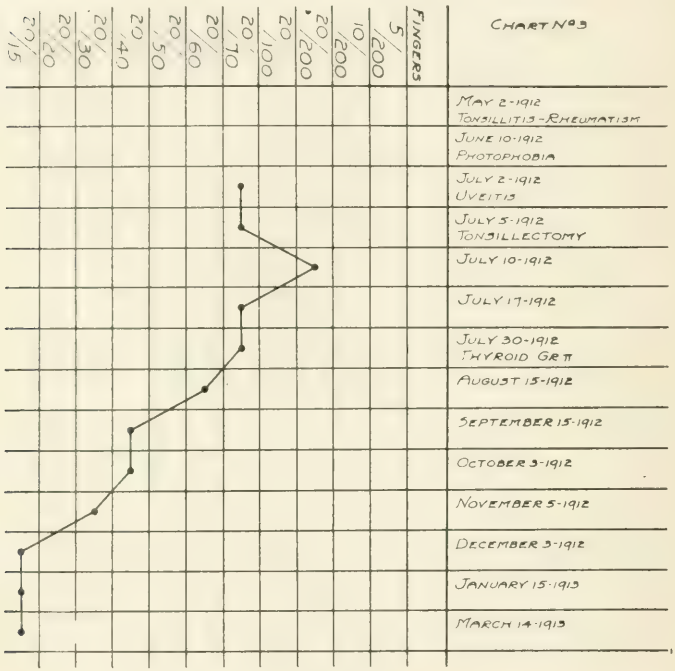


Chart 3.

but nothing was discovered except "suspicious tonsils." Wassermann test was negative and tuberculin reaction positive.

There were slight ptosis of the left upper lid, marked, deep circumcorneal injection, cysts in the conjunctiva, discoloration of the base of the iris, dots on the posterior corneal surface, semidilated and non-responsive pupil, and vitreous opacities.

She was placed in the hospital and her tonsils removed. Time was given to watch the effect of the operation. At first the vision and ocular condition became worse, then a reaction set in which resulted in an improved condition. When the vision had reached a point, and that far from normal, it became stationary, so twenty days after the operation thyroid extract was given. Following this there was a slow but gradual improvement until the vision became normal. The patient gained in weight and health, and now, two years later, is a robust woman, with nothing to show for her former eye trouble except a drooping lid.

CASE IV.—It is seldom that we see the first manifestations of congenital syphilis at the age of twenty-six, but in the following case such a condition obtained:

The patient, a rather frail, anemic woman, complaining of persistent photophobia of the left eye, came for a consultation. I could find nothing to account for her distress, nor could Dr. Thomas Futcher, to whom she was referred for a physical examination. She was kept under observation from March until July. During that time an abscessed tooth was extracted and a slight error of refraction was corrected.

On September 15, 1913, I found she had developed a definite uveitis of the left eye. There were exudates on the posterior surface of the cornea, circumcorneal injection, a cloudy vitreous, conjunctival cysts, a greenish discoloration of the base of the iris, and vision of $\frac{2}{40}$. She was given mercurial injections and iodid of potassium for four months, and, as there was no improvement in the ocular condition, pilocarpin was substituted and continued for two months. About March 1, 1914, she contracted what her physician, Dr. Albert Chatard, supposed to be influenza, which was quite prevalent in Baltimore at that time. She was confined to her bed for several days, and to her home for nearly two weeks. After convalescence a purulent nasal discharge persisted, and a small tumor of the left inferior turbinate, involving the floor of the nose, was discovered. She was referred to Dr. Keidel for Wassermann test, which proved positive. After blood of the husband and baby was tested and proved negative, the mother was confronted with the facts and confessed that her husband, whom she had divorced,

infected her with syphilis three months prior to our patient's birth.

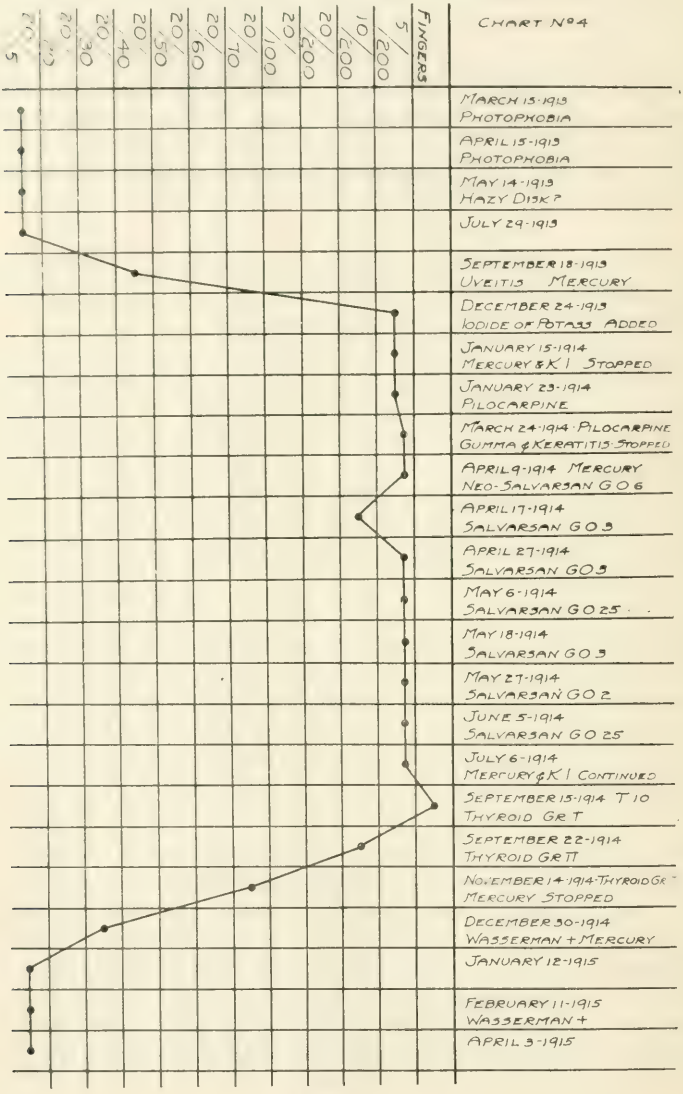


Chart 4.

At the time the gumma, which the nasal tumor proved to be, was discovered, a slight cloud was noted in the interstitial tissue in the upper portion of the cornea. In spite of salvarsan and mercurial injections, the corneal condition developed into a well-defined luetic interstitial keratitis. The vitreous became more cloudy, and the iris much inflamed.

She was given seven injections of salvarsan between April and June, 1914. Her nasal condition cleared up rapidly, but her eye symptoms persisted. Dr. Keidel was then obliged to discontinue the salvarsan "because of severe immediate reaction of so-called anaphylactic type." Mercury, however, was persisted in, as the Wassermann reaction remained positive.

On September 15, 1914, the patient was still suffering acutely, and the interstitial keratitis was no better. At this juncture she was given thyroid extract, starting with one grain a day. Within a week there was a marked change for the better, and, as she tolerated the thyroid perfectly, the amount given was increased to two grains a day. The subsequent improvement in her vision and ocular disease was so consistent and rapid that I feel it must be attributed to the thyroid extract. Certainly it was not salvarsan nor mercury alone which resulted in her recovery, because, while the Wassermann remained persistently positive, the vitreous cleared and the vision returned to $\frac{20}{15}$.

This case suffered both from uveitis and interstitial keratitis; nevertheless, I feel it can be classed with the other cases which form the basis for this communication.

CASE V.—Woman, aged twenty-four years, sent by Dr. James S. McLester to Dr. W. S. Thayer, and by him referred to me. To quote from Dr. McLester's history of the case: "At six or seven years of age Mrs. C. had an enlargement beneath the angle of her jaw believed to be sublingual adenitis. At thirteen, while having dental work done, a rather alarming cervical adenitis involving the glands of both sides appeared.

"The patient has been married two and a half years and has had two miscarriages. This was believed to be due to some uterine disorder.

"In the latter part of March, 1914, the patient became languid and had some fever. April 6th there appeared a swelling in the parotid region of either side. There was little pain, but fever, and she remained in bed about six weeks. About two weeks after the appearance of the adenitis double conjunctivitis was noted, and a little later marked dilatation of the left pupil. She was examined by an oculist who noted double-sided retinitis. At first the vitreous was clouded,

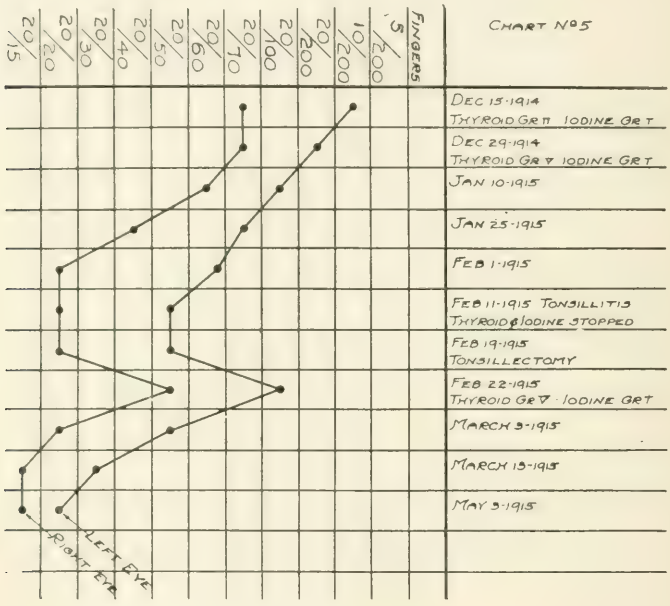


Chart 5.

but this later cleared up. She lost considerable weight during this illness.

"On June 28th the lungs were negative, as was the heart. The blood-pressure was normal. She was seen about this time by another oculist, who reported double neuroretinitis and was strongly inclined to regard this as luetic. Her hemoglobin was 70 per cent.; the leukocytes were 9500. The large number of eosinophiles suggested Hodgkin's disease. The feces were examined for ova, with negative results. The

urine showed albumin, but no casts. A phthalein test gave normal values.

"A Wassermann test was negative. She was given mercury hypodermically and another Wassermann test done, which also was negative. In spite of the negative Wassermann, we gave Mrs. C. mercury. She improved distinctly for a while, though the fever never disappeared, and we were never entirely satisfied regarding her eyes.

"Found two badly diseased molars. These were removed and distinct but incomplete improvement followed.

"Dr. Bunting could not be reached, and some smears were sent to Dr. Yates, . . . who wrote: 'Suggestion of Hodgkin's, provided tuberculosis and acute adenitis could be excluded.'

"An enlarged gland just above the clavicle appeared, was removed, and sent to Dr. Bunting. He wrote: 'The condition is one of chronic hyperplastic tuberculosis.'

"She was sent to visit her mother in Mississippi, and hygienic measures advised. On her return a few days ago we found the general condition satisfactory, but the eyes no better, particularly in view of a rather large exudate in the anterior chamber. I advised tuberculin therapy.

"One thing I do not feel entirely satisfied about: her teeth probably play a rôle in this, and we have not followed up this clue as persistently as might have been done."

I have nothing to add to this history, so thoroughly written by Dr. McLester. I might supplement his statement by saying that repeated Wassermann tests were in our hands negative and tuberculin tests positive.

The eyes were both involved in what we consider an undoubted malignant uveitis, as there were circumcorneal injection, exudates on Descemet's membrane, semidilated and non-responsive pupils, the bases of the irides were of a greenish color, and the vitreous filled with opacities. V. L. E. = $\frac{10}{200}$; R. E. = $\frac{20}{70}$. The fields were normal and tension was 15 in each eye. She suffered very much from photophobia, and the eyeballs were painful to the touch.

Dr. Thayer confirmed the report of anemia and suggested that we look for possible points of infection.

The patient was given thyroid—one grain a day gradually increased to four grains. She was, at first, given occasional doses of pilocarpin and hot-pack sweat-baths. Her improvement, both physical and ocular, was prompt and steady until the development of a mild follicular tonsillitis brought it to a stop. Still even during this infection there was no loss.

As soon as was thought safe the tonsils were removed by dissection. At first this appeared a tragedy from an ocular standpoint: her vision greatly declined, the photophobia and pain increased, and the exudates on the cornea greatly multiplied. After a lapse of three days, however, the thyroid, which had been discontinued, was renewed, and from that time on there was rapid improvement in the disease until the vision became normal.

Some of the dots on Descemet's membrane in the left eye persisted for several weeks, and a systematic examination of her teeth was made by Dr. C. J. Grieves, who reported: "One tooth, the first lower right molar, with a gold cap, has a blind abscess and should be extracted." After the extraction of this tooth there was a decided decrease in the descemetic dots in the left eye, and, in a letter just received from the patient, she says: "My eyes are much stronger and can stand the light, look natural, and my vision is perfect. I have gained about 30 pounds."

The only drugs given this patient were thyroid extract and iron. She could not tolerate atropin.

DISCUSSION.

Dr. Walter B. Weidler, New York: I should like to ask Dr. Bordley what method he used in making his tuberculin test—whether he employed the von Pirquet or made subcutaneous injections. I have done considerable work

with tuberculin for years at the Manhattan Eye and Ear Hospital, and had one specially interesting case of malignant uveitis in a woman thirty-five years of age. She had had the affected eye enucleated at the Mt. Sinai Hospital on account of uveitis. Dr. Shoenberg had seen the patient there, and had performed the enucleation. He told me that he had found a history of syphilis. When the woman came under observation at the Manhattan, I found, on examination, that her vision in the remaining eye was $\frac{20}{50}$. This gradually decreased until it was $\frac{20}{100}$. The Wassermann test was negative, and her history was negative as to syphilis. A von Pirquet was positive and a subcutaneous injection of tuberculin was made, and we secured a general reaction, with a rise of temperature, and a focal reaction in the eye. The patient was put under treatment with injections of tuberculin emulsion, the treatment being continued for two months or more. There was a gradual improvement in the condition in the eye, no other form of treatment having been used during this time. The uveitis entirely cleared up, and she left the hospital with a vision of $\frac{20}{15}$.

I was interested in the case for the reason that the left eye had been previously removed for an apparently similar condition, and I almost fancied that the left eye might have been saved had it been treated in a similar manner. I think it was in the second case that Dr. Bordley reported in which tuberculin had been used for a certain length of time, and I should like to ask how long he used it, and in how large doses, before he changed the treatment to iodids.

DR. G. O. RING, Philadelphia: I should like to ask Dr. Bordley about the dosage of thyroid that he used.

DR. ROBERT L. RANDOLPH, Baltimore, Md.: I am particularly desirous to know whether Dr. Bordley has discovered in the *early history* of uveitis any feature which establishes the fact that the disease is malignant and not amenable to the usual methods of treatment.

DR. SAMUEL D. RISLEY, Philadelphia: It occurred to me, while Dr. Bordley was reading his paper, that many of our patients with interstitial keratitis may properly be classi-

fied as cases of malignant uveitis. A few years ago, before this Society, I presented a brief paper on the etiologic factors in interstitial keratitis, in which the claim was made that whether the corneal disease was due to inherited or acquired syphilis, or occurred in the children of unfit parentage, or in those whose early nutrition had been impaired by the evil circumstances under which they had lived, the interstitial keratitis was quite uniformly associated with these conditions, or any others that might impair the general vitality or nutrition of the individual, and more or less directly dependent on them; therefore that interstitial keratitis was not necessarily luetic in origin, and reported cases to substantiate this claim, but maintained that the sequelæ of the general impairment of nutrition did not fall alone upon the avascular structures, like the cornea, but also on the thyroid gland, thus weakening the function of the thyroid in maintaining the general nutrition. Cases were reported in that paper which had failed to recover or improve under the usual methods of treatment adopted for interstitial keratitis, but had recovered rapidly under the use of pulverized thyroid gland of the sheep. My thought, as set forth in that paper, was that all that was needed for these patients to recover from this malignant condition was the restoration of the function of the thyroid, which was aided by the administration of the gland. In cases of keratitis of the interstitial type, associated as they frequently are with malignant uveitis, their malignancy may have in large measure depended on the condition that I have so briefly tried to outline.

Dr. William K. Rogers, Columbus, Ohio: I would like to ask Dr. Bordley whether he considers the administration of thyroid necessary after apparent subsidence of the ocular symptoms, or whether it is simply indicated during the continuance of the active inflammatory condition.

Dr. James Bordley, Jr., Baltimore, Md.: In answer to the question of Dr. Weidler in regard to the amount of tuberculin used in the first case and the length of time it was used, I would state that I am sorry to say that I am unprepared to tell the dosage. The doses were small and were given by Dr. Hamman, who is in charge of the Department

of Tuberculosis in Johns Hopkins Hospital. They were given from May until July. During that time the patient's vision fell rapidly, and the treatment was changed.

The dosage of thyroid extract depends very largely on the individual. With some patients we give as much as five grains twice a day; with others, half a grain or a grain twice a day would produce palpitation. The only way this can be determined is by testing it in the case of each individual.

These reported cases differ from those of ordinary uveitis. The condition is malignant in that it is absolutely progressive. I have seen eight well-marked cases of it. In spite of all treatment there is no remission in the symptoms—unless something is done to stop the process it will blind the eye. I differentiate it from the ordinary form by observing that there are cysts in the conjunctiva, there is atrophy at the base of the iris, and the manner in which the vitreous opacities form. They start at the top of the chamber and hang like a veil, which gradually lengthens until it fills the vitreous chamber.

In regard to the length of time that I give the thyroid, I would say that I usually continue it until vision is normal, or has reached what I consider to be normal vision for that eye. In one case we kept treatment up for six months, so as to see whether we could not better the vision beyond $\frac{20}{30}$, but we could not do so. I have a patient on thyroid now, of whom I did not have time to speak. Her vision has been normal since March. She is still on thyroid because she is vastly improved in health by its use. She has increased in weight, and her physician, a very competent man, rather insists that we do not stop the treatment as long as her physical condition remains normal. I should think, however, that the vision would be a good index, because, as the patient becomes normal, the pathologic process ceases and the vision improves.

PROGRESSIVE ESSENTIAL ATROPHY OF ALL LAYERS OF THE IRIS.

G. E. DE SCHWEINITZ, M.D.,
Philadelphia, Pa.

With iris atrophy manifesting itself in depigmentation, thinning of its substance, disappearance of the stroma cells, fibrous transformation, thickening and hyaline change of the blood-vessels, we are all familiar. The lightest degree of this phenomenon is seen in the diffuse atrophy which underlies the so-called heterochromia; more pronounced phases may be the sequel of various types of inflammation, as this occurs in recurring iritis and infective iridocyclitis. A common type of iris atrophy arises in consequence of increase of intraocular tension, and depends upon compression of the blood-vessels of the root of the iris, and perhaps upon compression of the ciliary nerves. As has been emphasized by Hirschberg, it may arise rapidly, even if the increased pressure has lasted only a day or two. Other frequently encountered types of iris atrophy follow rupture of the vessels which run from the major arterial circle, as, for example, in iridodialysis, or traction of the iris tissue, for instance, after its incarceration in corneal scars. Local iris atrophies are caused by the absorption of tuberculous and syphilitic lesions, or may develop in the neighborhood of synechiæ; more diffuse atrophies occur in connection with siderosis, and extensive, even total (Stoewer) ones after traumatic hemorrhage. Certain atrophies referred to by Axenfeld depend upon hyaline degeneration of the pupil margin, and other types eventuate in complete loss of the iris plications and are encountered in locomotor ataxia and

paretic dementia, and depend, according to Dupuy-Dutemps, who has written concerning them, upon trophic disturbances.

The appearance of atrophy of the iris which follows inflammation and increased intraocular tension has been aptly compared by Fuchs to that of gray blotting-paper. Should the atrophy be circumscribed, he continues, and the stroma pigment be destroyed, light and even white spots occur, and if they are numerous, the condition develops which has been described under the name *vitiligo iridis*. A still further progress of this spotty atrophy of the iris consists in a loss of its stroma, so that the retinal pigment layer is exposed, and if this too is destroyed, gap-formation is evident.

With these various types of iris atrophy the present communication is not especially concerned, inasmuch as I shall attempt to describe what appears to have been an essential progressive atrophy of all the layers of the iris, not, however, entirely unassociated with some of the conditions, even though in minor degree active, which, in general terms, are responsible for the usual atrophic changes in this portion of the uveal tract.

History.—The patient is an unmarried woman, aged twenty-three, whose parents are living and in good health. She is the third child in the family, and has two brothers and two sisters, living and well. Two maternal uncles had nervous prostration: one recovered entirely, the other has not regained complete health. The patient has had measles, scarlet fever, whooping-cough, chicken-pox, and pneumonia. Her birth was normal, but she was a delicate child, and her mother, before this daughter was born, in general terms was in poor condition, and when two months pregnant had numerous fainting spells and some form of "slow fever," which may have been malaria, although there is no record of blood examination to prove whether the malarial organisms were present or not. In childhood the patient readily

"caught cold," especially in the winter time. At twelve years of age she had lobar pneumonia, and four years later pleurisy. At this time, or a little earlier, she was thrown from a wagon which collided with an automobile, injuring her left side and causing a high degree of shock, and while attending school she had adenitis of the neck and suppuration of the axillary glands.

The patient left school when she was eighteen years of age, much improved in general health. Soon afterward her mother's illness compelled the patient to help her father in his business in bookkeeping, and to spend much time in attending to household duties, for which her mother was unable to be responsible. Consequently she became weak and tired, and began to notice the condition of her eye which shall presently be described; that is to say, the ocular lesions first made their appearance in the early portion of 1912.*

In general terms the patient has frequently suffered from headaches on both sides of the brow, particularly when exposed to the light, but not alone excited by the action of light. Hearing is good, but especially during fits of nervousness there is a sense of fulness in the head. Sharp pains have from time to time appeared in the chest, and there have been periods of tachycardia. Her menstrual periods were established at fifteen years of age, and the duration of the period is usually from six to seven days, associated often with abdominal pain. The pelvis has been examined, but with the exception of a slight uterine displacement, no lesion of any material importance has been discovered. Vague rheumatic pains have at times attacked her hips, but there is no evidence of arthritis of any kind or description. Teeth and nasopharynx are in good condition; the appetite is healthy, and her food habits are normal.

In January, 1913, therefore about a year after the first iris changes were noticed, about eight o'clock in the evening, the patient, after a sensation of coldness and drowsiness which had lasted all day, experienced numbness in the fingers of the right hand and tingling as if being pricked with pins and needles, and a sensation as if electric shocks were passing through the hips and right leg. This tingling sensa-

* The patient states positively that her eyes were examined by an oculist during her school days, and that they were entirely normal.

tion continued, was unrelieved by the usual remedies, and by the end of the third day the patient had failed to regain full power of her right hand, and on the following day she was hyperesthetic and unable to bear the touch of even the lightest bedclothes. While her arm could be moved, she had no power in it, and was obliged to be bedridden for four or five weeks, but the hand did not regain its normal power for about two months. Even at this period the tingling did not entirely cease, but was reëstablished if the hand touched anything. Gradually the sensation disappeared and comparatively normal strength returned.

Dr. D. J. McCarthy was called in consultation by her family physician, Dr. Councill, and writes thus concerning his investigations:

"I have made a careful examination of your patient, and was not able to detect any evidence of organic disease of the central or peripheral nervous system; station is good; gait is normal; reflexes are normal; muscular power is normal. There is no atrophy or wasting, and the only disturbance of sensation is slight hyperesthesia to pin-point in the right leg, which I think can be attributed to a functional disturbance. As to the series of functional nervous disturbances in this case, it would appear, from the history of pneumonia, pleurisy, suppuration of the axillary glands, etc., together with the presence of a slight enlargement of the post-cervical glands at the present time, that we might be dealing with a latent tuberculous infection causing the drag on the nervous system. I was not able to detect definite lesion in the lungs, although there is a slight impairment of resonance over the right apex, anteriorly, evidently due to the old pleural or pneumonic condition.

"There is a well-defined Chvostek symptom (hyperirritation of the seventh nerve to percussion), which, in the absence of tetany, is practically always an evidence of some intoxication, and most frequently an intoxication from the gastro-intestinal tract. I was not able, however, to find anything from a physical examination to indicate any disturbance in this area.* The blood-pressure apparatus recorded: systolic, 120; diastolic, 80 (Stanton apparatus).

* A complete metabolic study is to be made, but could not be furnished in time for this report.

As a whole, the case rather impresses me as one of low nerve tone due to a latent glandular tuberculosis with chronic intoxication."

Eye History.—The earliest ocular examinations were made by Dr. Frank Fisher, who kindly furnishes the following data: The patient was first examined March 19, 1912. She came with the statement that, for a month or more, certain changes had been noticed in the appearance of the left iris, and in response to questions gave the history of the accident referred to earlier in this paper, to wit, that eight or nine years prior to this examination she had suffered much from shock as the result of an automobile accident. There appeared to be a slight proptosis of the left eye, and the refractive error was corrected by −0.50 cyl., axis 180 (the vision is not given). The iris had the appearance of degeneration

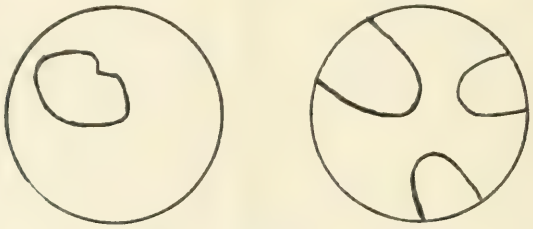


Fig. 1.—Appearances of iris March, 1912, and June, 1913.

and thinning in spots, the pupil being egg-shaped, with its point up and in. There was a small gap at the extreme outer margin near the base. In August the pupil had become more ovoid, and a second fenestrum had formed above the first described. In January, 1913, there was an increase in the size of all the holes and in the width of the pupil. The appearances diagrammatically depicted by Dr. Fisher, as they were noted in March, 1912, and again in June, 1913, are exhibited in Fig. 1.

The patient was first examined by me on July 9, 1913. The history previously recorded was extracted, and in addition to the examinations there noted her physician, Dr. Councill, reports that the Wassermann test was negative. V. O. D. + 0.25 cyl., axis 90 = $\frac{6}{5}$; O. S. −0.25 sph. ⊂ − 0.37 cyl., axis 180 = $\frac{6}{6}$.

Fig. 2.—Appearance of iris July 9, 1913.

Fig. 3.—Appearance of iris January 22, 1915.

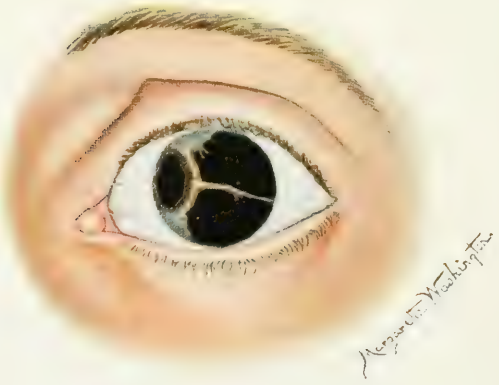


Fig. 4.—Appearance of iris May 5, 1915.

The eye-ground contained no lesions, and the field of vision was perfectly normal. The appearances of the iris are shown in the water color by Miss Washington (Fig. 2); to wit, an oval pupil, pointing slightly upward and inward, the entire iris thin, of a somewhat gray-blue color, and, on the outer side, extending to the limbus, two large and one small aperture, the largest of the three being crossed by a single strand of iris. Gradually the ovoid pupil previously described changed in shape and was more displaced, until it reached practically the margin on the nasal side and became vertically oval in shape. The three fenestra noted in the previous sketch had in the mean time coalesced, and now had become one large gap, crossed by an irregular band of iris practically in the midline, and by January 22, 1915, the appearances were those seen in the water color by Miss Washington (Fig. 3).

The first definite rise of tension noted while the patient was under my care occurred on April 20, 1914. The tension, however, was tested with the finger and not with the tonometer. After three days' use of pilocarpin, $\frac{1}{10}$ grain to the ounce, the finger tension became normal, and the pain which had accompanied this rise of tension subsided. One month later for a period of two days the eye was blood-shot, and there was some pain over the eyebrow and occiput. There was also a slight bulbar injection, but no synechiæ formed, and there was no failure of the distorted pupil to react to light. Again the finger tension was above the normal and all the symptoms disappeared after a few days' use of dionin and pilocarpin. Curiously enough, a few days later a precisely similar flush appeared in the right eye, but there was no rise of tension, and during this period of time there was no fundus change, and the disc and vascular distribution in the retina were perfectly normal.

During the month of January, 1915, although finger palpation appeared to indicate a slight rise in tension, the tonometric measurement was never above 25. During the following three months several slight scleral flushes were evident, unassociated with the discoloration of the iris or the formation of synechiæ, and on one of these occasions this flushing of the sclera, not, however, localized in the ciliary zone, lasted for a week, and during this period of time there

was slight nausea and vomiting. In June of the present year, when the last examinations were made, the tonometer tension was 21, but the patient has used continuously for a long period of time a weak solution of pilocarpin about $\frac{1}{10}$ grain to the ounce. The right eye has a grayish-blue iris, with a distinct tinge of yellow in the minor circle. In the left eye and those portions of the iris remaining a slight tremulousness on motion was noticeable and the edge of the lens could be distinctly seen. The color of the strands of iris which still exist is more gray than blue, and the yellowish tinge is greater than in the other eye. The inner nasal opening, which represents the original pupil, is surrounded by a yellowish border which constitutes the remnant of the minor circle, and there is a well-marked band of ectropion of the uveal pigment. This pupil exhibits in diminished degree the usual reactions. The vision at the present time, June 12, 1915, is: O. D. + 0.50 cyl., axis 150 = $\frac{6}{5}$; O. S. − 1 sph. ⊂ − 1.25 cyl., axis 150 = $\frac{6}{9}$.

The visual field was not far from normal for colors, with a slight concentric contraction on both sides, but a little greater on the left side, and with the contraction slightly more pronounced on the nasal half than elsewhere. There is no cupping of the disc and no pulsation of the vessels, and the flushings of the sclera previously described have not been repeated. The patient's general condition is very good, and since the last glasses have been adjusted, the headaches from which she suffered from time to time have practically disappeared. The iris at this time (May, 1915) is depicted in the water color by Miss Margaretta Washington (Fig. 4.) This patient was also examined by Dr. John T. Carpenter in July, 1913, and conditions similar to those depicted in Fig. 2 were evident.

Remarks.—While it has not been possible to make an adequate examination of the literature of this subject, one or two communications may now be referred to. Casey Wood describes a progressive primary atrophy and almost complete disappearance of the right iris in a man aged forty-four. This patient sustained a fracture of the right superior maxilla in 1891, and in 1907 and 1908 exploration of the antrum dis-

covered pus, which was attributed to influenza and not to the injury previously described. The atrophy of the iris began in 1904, and first manifested itself in what appeared to be a gradual enlargement of the pupil, eventuating in a gradual disappearance of the iris. These phenomena were watched by Wood from 1907 until 1909, and there was a steady progressive disappearance of the iris stroma, unaccompanied by any symptom except increased tension. It should be noted, however, that the patient before he saw Wood had periods of photophobia and wore smoked glasses. By the beginning of 1909 fully $\frac{5}{6}$ of the iris tissue had disappeared, and the pupil had assumed the shape of an irregular triangle. The absorption of the iris tissue began on the anterior surface; gradually glaucoma developed, and the visual field, originally normal in extent, began to contract. Cyclodialysis and miotics furnished no relief. Toward the end of 1909 the eye was so painful and congested that it was removed by the surgeon who then had the patient in charge and sent to Dr. Wood for examination. The findings are thus summarized: Subacute and chronic fibrinoplastic iridocyclitis; secondary glaucoma; extensive secondary atrophy of the entire iris. Wood concludes that spontaneous atrophy of the iris has a varied pathogenesis, but thinks it likely that every example of the disease would be found to exhibit toward the end of the process more or less marked glaucoma, which is probably an incident in the disease, and depends upon an effort of the eye to rid itself of the irritating products of the primary affection.

He refers to G. Lindsay Johnson's case of progressive atrophy of the left iris in a patient who had lost his right eye from injury and who had always been healthy. This atrophy was watched by Johnson for seven months, and at the expiration of that time only a small band of iris above and below remained. The optic disc and macula were unaffected, and the fundus immediately around it was healthy; else-

where there was atrophy of the choroid. No explanation is given, and there is no record of increased tension.

He refers particularly to a case of atrophy of the iris which occurred without the influence of mechanical injury reported by Harms, the patient being a healthy man whose general condition was normal, but who, when examined by Harms, had well-marked simple chronic glaucoma. Wood agrees with Harms that it is possible that if their patients had been examined before the iritic atrophy had advanced the eye would have been found without glaucomatous signs. In other words, the end-process of the disease is a quiet secondary glaucoma.

The case which most nearly resembles the one which I have reported has been briefly recorded by William Zentmayer, and is described by him as essential atrophy of the iris, which occurred in the left eye of a woman aged twenty-seven years, who stated that eleven months prior to her examination by Zentmayer she had noticed that the dark part of the left eye was increasing in size. She gave no history of trauma or inflammation, although she stated that the eye became injected after prolonged near work. The pupil at the time of examination was 7 by 4 mm., egg-shaped, pointing upward and outward, reaching to within $1\frac{1}{2}$ mm. of the corneal margin. There were half a dozen fenestra in the nasal part of the iris, extending from the ciliary border to the smaller circle. The pupil reacted to light. Tension by palpation was + ?. Through the fenestra the suspensory fibers could be seen, and the fundus was normal. Eight months later the entire nasal third of the iris had disappeared except for the sphincter and a few connective-tissue strands stretched between the sphincter and the ciliary attachment. Zentmayer suggests that an explanation of the condition resides in a sclerosis of the vessels of the iris leading to atrophy of the stroma from loss of nutrition. There is no

record of any general examinations, nor is the cause of this suggested sclerosis of the vessels of the iris described.

In the present instance it is hardly possible to believe that the automobile accident which occurred long before the iris atrophy began has any relationship to this progressive disappearance of the iris stroma. The general examinations certainly indicate very definitely the presence of a latent tuberculous infection, and almost certainly a gastro-intestinal intoxication. This would furnish a probable constitutional basis for the condition. Certainly during the process of the atrophy there have been periods of rise of intraocular pressure, if finger palpation may be trusted, although tonometric measurements have never demonstrated a tension above the high physiologic limit, but it must be noted that the tonometer was not used until after pilocarpin had been employed.* That there is some process in the anterior uveal tract which interprets itself in periods of flushed sclera, pain, tenderness, and even, on one occasion, sufficient discomfort to cause nausea and vomiting, is evident from the history and the examination. So, too, in Wood's patient there is a history of photophobia and a period of wearing dark glasses.

Now it is conceivable that these phenomena are interpretations of vascular changes to which Zentmayer makes reference, and that this vascular disease, if I may so express myself, is the immediate exciting cause of the progressive atrophy, just as we know that pressure upon the vessels of the major circle of the iris, as Fuchs points out, causes atrophy of its structure, for example, in various types of glaucoma, aided, it is quite possible, by disorders of innervation, for example, compression of the ciliary nerves, exactly as Dupuy-Dutemps summons this etiologic factor to explain the marginal atrophies which he has noted in tabes dorsalis.

*Since this sentence was written the patient, in September, 1915, had a sharp rise of intraocular pressure, 35 + mm., and during its presence there was rapid pulsation of the central vein, which ceased when the pressure was reduced by active miotics.

It will be remembered that Galezowski tried to explain certain types of diffuse atrophy of the iris as they are seen in heterochromia as the result of paralysis of the sympathetic on the same side, an explanation which he himself rejects, inasmuch as in other cases the same state of affairs occurred when there were signs of excitation instead of paralysis on the side of the depigmented iris.

Evidently, in this patient at least, as I have already pointed out, two insistent underlying constitutional conditions are present—a degraded nervous control, probably dependent upon latent tuberculous infection, and certainly in one period of the patient's life, at least, gastro-intestinal intoxication. From these sources it is fair to assume that some toxin may have arisen which has excited a vascular change, which in turn has encouraged the melting away of the iris stroma. We know that even in so serious a business as peptic ulcer the underlying pathologic state is badly understood, although usually attributed to vascular disturbance, thrombosis, or embolism, but undoubtedly also to a recurring vascular spasm which inhibits the nutrition of the area involved. One might also speculate on the relationship of the nerves to this condition, exactly as I have already mentioned, quoting from Dupuy-Dutemps, and the process perhaps might be, in part, described as a neurotrophic atrophy, such as occurs in the skin in the presence of neuritis and pressure on the supplying nerve plexus. That rises of intraocular pressure become an important part of the phenomena which have been described is certainly true, especially as described by Wood and Harms, and in Zentmayer's patient, as well as my own, rises of tension have certainly been demonstrated, but whether these are part of the process which has excited and caused the atrophy, exactly as glaucoma causes atrophy of the iris when it is of long standing, or whether they are only an incident in the disease, to quote Casey Wood's expression,

and depend upon an effort of the eye to get rid of the irritating products of the primary affection, is not plain.

Finally, as a rather far-fetched theory, it may be suggested that there is some fault in the secretion of ductless glands. We all know that parathyroids, for example, have some very distinct relation to calcium metabolism, and that their removal may be followed by unusual symptoms, notably by tetany. Perhaps in similar manner some disturbance of internal secretion may be followed by local spasms in the muscular layer of the iris, the repetition of which is followed by atrophy, exactly as these disturbances of internal secretion are followed by the tonic cramps in certain voluntary muscle areas, and permanent contractures occur, as they do in tetany. All this is wandering far afield without any good foundation on which to base the theories, and I must return to the original statement that the exact or even likely cause of the condition which has been described is not yet discovered.

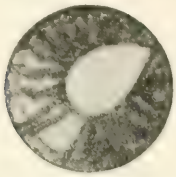
REFERENCES.

Axenfeld: Bericht über Versammlung der Ophth. Gesell., Heidelberg, 1911, p. 255.
Dupuy-Dutemps: *Annales d'Oculistique,* September, 1905.
Fuchs: Text-book of Ophthalmology, translated by Duane, fourth edition, 1911, p. 397.
Harms: *Klin. Monatsbl. f. Augenheilk.,* November–December, 1903, p. 522.
Hirschberg: *Centralbl. f. prakt. Augenheilk.,* June, 1907.
Johnson: *Ophthalmic Review,* 1886, vol. v, p. 57.
Stoewer: *Klin. Monatsbl. f. Augenheilk.,* February, 1904.
Wood: The Ophthalmoscope, 1910, vol. viii, p. 858.
Zentmayer: *Transactions of the College of Physicians,* third series, 1913, vol. xxxv, p. 456.

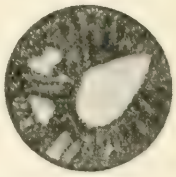
DISCUSSION.

Dr. William Zentmayer, Philadelphia: There is no necessity for me to go into the details of my case, as Dr. de Schweinitz has described it fully, but I should like to exhibit this rough sketch, which shows that between May and December the atrophy had increased to such an extent that the fenestra had coalesced, leaving an atrophic area involving the nasal half of the membrane. In regard to the prob-

able cause of it, we may theorize, but we can come to no definite conclusion.



Left eye, May, 1913.



Left eye, December, 1913.

Dr. W. F. Mittendorf, New York: The case of atrophy of the iris that I should like to report occurred in a young army officer. I had been attending this young man at the age of ten or twelve years, when he had had repeated attacks of rheumatic iritis. These passed away, and he was able to pass the examination for admission to West Point. Later he graduated and went to the Philippines, where he stayed for several years. On his return he came to me for blurring of the sight and sensitiveness to light. I found that the entire iris had disappeared, leaving only little bands running from the pupil to the periphery. The condition presented the appearance of polycoria in a marked degree. Vision had dropped from $\frac{20}{50}$ to $\frac{20}{40}$. Later he returned to the Philippines, where he had an attack of acute rheumatism and died.

Dr. Edward Stieren, Pittsburgh, Pa.: At the meeting of the Pennsylvania Medical Society in Pittsburgh last September I exhibited a patient with atrophy of the iris. I was fortunate in having Dr. de Schweinitz examine this case.

Briefly stated, the condition was one of high myopia, with increased tension, in a stenographer twenty-three years old, with repeated attacks of bullous keratitis and a coloboma of the iris, with several areas of thinning in the iris.

The coloboma developed slowly, in about a year's time, and followed the appearance of a yellow spot on the iris. There had never been any pain or inflammation associated with the condition. Tuberculin reaction was negative, as was also the Wassermann test. Dr. de Schweinitz gave

as his opinion that the yellow spot on the iris may have been exudation from a localized fibrinoplastic cyclitis, the disappearance of the exudate being followed by the atrophy of the iris.

As a relief measure from the high intraocular tension, which at times would be 45 mm., and also to secure a section of the iris for microscopic study, an iridectomy was attempted but failed, as the iris was so friable that all efforts to draw it through the corneal section resulted only in tearing it to shreds. Later the eye was trephined with success. The tension now remains low, no further attacks of bullous keratitis have occurred, and the patient is able to follow her vocation with comfort.

Dr. Samuel D. Risley, Philadelphia: I have under observation at the Wills Hospital at this time a case of polycoria which I should like to add to those already mentioned.

Dr. Robert S. Lamb, Washington, D. C.: Was the occurrence of pain in the case reported by Dr. de Schweinitz intermittent, or was there definite pain in the eye?

Dr. de Schweinitz: Replying to the question of Dr. Lamb, I may say that there were only three periods while the young woman was under my care in which there was any pain. In each instance except one the pain was slight, and lasted only from twenty-four to thirty-six hours, being relieved by pilocarpin. On one occasion, however, it lasted over a week, and was severe enough to produce nausea and vomiting. There was no change in the iris itself. All tests were negative except the skin-tuberculin test, which was positive. No internal remedies have been tried.

THE OPHTHALMIAS OF PALESTINE.

HARRY FRIEDENWALD, M.D.,
Baltimore, Md.

A journey made four years ago to Egypt and Palestine brought to my attention, as it does to every traveler in these countries, the prevalence of conjunctival diseases and the frequency of blindness. But as these observations were necessarily superficial, I determined to study them more carefully on another visit. Such an opportunity offered itself last summer. Provided with all the necessary instruments compactly packed, and with the assurance of all possible aid from a number of the leading physicians* in Palestine, I spent six weeks in various cities, towns, and villages and examined a large number of patients and the pupils of numerous schools. It is on the basis of this experience that I present the following report:

Egypt.—The subject of the ophthalmias of Palestine cannot be approached without a brief consideration of the ophthalmias of Egypt. The frequency of the occurrence of ophthalmia in the latter country is long known; it has been written about in ancient as in modern times, and affords ample explanation for the name "Egyptian ophthalmia." The bibliography of ophthalmic disease in Egypt is very extensive. Hirschberg, in particular, has discussed the subject in a very interesting manner; and the interesting work done by Dr. Howe on the transmission of ophthalmia through the fly

* I take pleasure in expressing my deep gratitude to Dr. Segal (Rothschild Hospital, Jerusalem), Dr. Ticho (Jewish Ophthalmic Hospital, Jerusalem), Drs. Heron and Thompson (British Ophthalmic Hospital, Jerusalem), Dr. Feigenbaum (Jewish Health Bureau, Jerusalem), Dr. Auerbach (Haifa), Dr. Muscovitz (Rehobeth), and Dr. David (Yemma).

is known to you. The most recent monograph, entitled "Trachoma and its Complications in Egypt," by Dr. A. F. MacCallan, the director of the ophthalmic hospitals of Egypt, gives an excellent summary of the subject. He makes a rapid survey of the historic references, beginning with the "Ebers Papyrus," but bases his description of conditions chiefly upon the very extensive experience of the British ophthalmic hospitals, established under his direction in 1904, and rapidly increasing in number. He tells us that trachoma affects 95 per cent. of the population of Egypt and all localities and latitudes; no race is immune. Children are generally affected during the first two years of life, and the occurrence of ophthalmia neonatorum trachomatosa is unquestioned. Uncleanliness is the chief cause of its wide distribution; he regards flies as the least common means of transmission.

Among the complications of trachoma in Egypt acute ophthalmias, and among these gonococcic conjunctivitis, are not uncommon, and the resulting inflammation is quite different from anything with which we are familiar in Europe or America. Gonococcic conjunctivitis is usually transmitted from eye to eye. "The presence of trachoma predisposes the conjunctiva to infection by gonococci. The presence of pannus to some extent protects the cornea from its destructive effects."

The association of vernal conjunctivitis with trachoma is not infrequent in Egypt; the palpebral form is the common one, and is almost always complicated with trachoma.

On the subject of blindness in Egypt, MacCallan reports on 23,000 blind eyes seen in five years. Sixty-nine per cent. of these cases were due to ophthalmia, and, by way of comparison, it is interesting to note that 14 per cent. were due to glaucoma, 4.5 per cent. to fundus disease, 4 per cent. to cataract, and 1.5 per cent. to injury. The census shows that there are more than 500,000 blind in one or both eyes in

Egypt, or 4.5 per cent. of the population, but the director general of the census states that this number is too low, and that the actual number is probably 1,000,000. MacCallan gives some interesting statistics based upon school examinations:

Of 2270 pupils of the infant schools of Tanta, there were:

Healthy	59
Conjunctivitis not definitely diagnosed	138
Trachoma	2073
Percentage of trachoma	91.32

It is also interesting to note that 65 pupils were blind in one eye and 21 blind in both eyes.

Of 969 similar pupils of Asuit, 34 were blind in one eye, and the percentage of trachoma was 97.5.

In the Tanta Primary School there were:

Number of pupils examined	969
Healthy	17
Trachoma	952
Percentage of trachoma	95.3

At this school the amount of trachoma has varied from 95 to 96 per cent. during the last five years, and this indicates the average percentage of the population infected with the disease.

In order to show the effect of trachoma upon the vision there is an interesting comparison between the visual tests of an Egyptian school, of the school children of Leith, and of a certain London county school; while in the two latter the pupils with normal vision ($\frac{6}{6}$ in each eye, with correction of ametropia up to + 6 D. or − 6 D.) numbered about 75 per cent., in the Egyptian school they furnished 18.8 per cent. Whereas the number who had vision $\frac{6}{18}$ or less in one or both eyes was 8.1 per cent. in London, 18.23 per cent. in Leith, it was 46.4 per cent. in Egypt.

"In England and Scotland the majority of the pupils have good vision, while in Egypt the majority have bad vision."

Corneal opacity was the cause of subnormal vision in 172 out of a total of 366 cases.

Meyerhoff, who has written extensively on ophthalmia in Egypt, and with whom I had the pleasure of a long conference while in Cairo, states that gonococcic conjunctivitis is endemic at present in Egypt, as it was from seventy to one hundred years ago in the European armies, and that it spreads in epidemic form annually from June to December, reaching the climax as regards both frequency and severity in September and October. Small children are most frequently attacked. Its spread is favored especially in the hot season by a chronic purulent conjunctivitis, which is no longer seen in Europe, and evidently clings to the trachomatous conjunctiva. Recovery without treatment is not rare, but from 35 per cent. to 50 per cent. of all patients show corneal involvement when they first present themselves for treatment. The rest are, for the most part, curable by irrigation or applications of nitrate of silver, the pannus acting, in a measure, as a protection to the cornea. But the gonococcic inflammation does not cure the trachoma, nor does the latter make the course of the former milder in any degree. Blennorrhea is exceedingly frequent in trachomatous conjunctivitis. Trachoma after blennorrhea as the result of simultaneous infection is not rare, especially in children. Mixed infections of gonococcic conjunctivitis with other forms of acute conjunctivitis are frequent.

Meyerhof shows that combinations of trachoma and vernal catarrh are frequent, but that the diagnosis of the latter is often difficult. It is commonly found that a case of trachoma is subsequently affected with vernal catarrh.

I have referred with some detail to ophthalmias of Egypt because of their similarity to those of Palestine. The essential difference, as we shall see, lies in the fact that Palestine ophthalmias are less severe, especially the acute forms. I may add here that I did not see in Palestine any cases of the "Egyptian florid trachoma" of Meyerhof, resulting from the infection of a trachomatous conjunctiva with gonococci.

PALESTINE.—The first study of trachoma in Palestine was made by Theodor Germann. He was sent by the Russian Palestine Society to examine into the condition of the eyes of

the school children, and to arrange for their medical and hygienic care. His mission was carried out between the tenth of September and the twenty-first of December, 1896.

He regarded the absence of rain, the hot and blinding sun, the chalky dust, uncleanliness, the scarcity of water, and poverty as the chief causes of the prevalence of ophthalmias. The fine, thorn-like hairs covering the fruit of the cactus, the juice of the fig, and flies are commonly looked upon as the causes of inflammation of the eyes and its distribution. Germann is not prepared to deny that these hairs do play some part in the causation of inflammations of the eye. He himself saw no case of injury from cactus. As to figs, he is inclined to believe that this fruit and others may be the means of transference of disease through the filth clinging to the hands. He ascribes the part played by the fly to the filthy hands in their effort to drive the fly away from the face. He seems to think that flies do not go from infected eyes to healthy ones, but rather from one infected eye to another, and that the part played by the fly in general is exaggerated.

He is of the opinion that the head-dress (fez) is responsible for the hands being very frequently carried to the face, because the eyes are not protected from the hot sun.

Germann's dispensary service embraced all sorts of people. He found trachoma and its sequelæ the most important disease and the catarrhal inflammations next. He is convinced that the ophthalmic conditions could be made very good, for the examination of the eyes of the 103 school children in the large German colony of Haifa did not reveal a single case of trachoma. He examined 2010 children in Syria and 1096 in Palestine and 1228 adults. He found conditions better in Syria than in Palestine, and observed catarrhs frequently chiefly in children, and especially in the younger years. He lays stress on the diminution of the frequency of the disease when the cold weather comes on, and quotes an Arabic proverb, "Green land clears the eyes," and points out that catarrhs prepare the field for subsequent trachoma. He saw no cases of blennorrhea. He states that he saw only the large granular chronic trachoma and no cases of acute trachoma or of follicular catarrh. He finds less trachoma in

Syria than in the north of Russia, but in Palestine more. The disease is otherwise similar to that in Russia, except that trachoma is seldom seen before the fifth year in the latter country, and is common in Palestine in very young children—even as young as one year. In these cases the mothers were always likewise affected.

Until he reached Jerusalem he was of the opinion that trachoma was not so serious an affection as he had been told. In Jerusalem he saw 540 patients; in Bethlehem, 367; in Nazareth, 321, making a total of 1128 cases, and among these there were only 37 cases in which there was not a mixture of various stages of trachoma. His statistical tables show the following:

	Per Cent.
Syrian schools	66.32 healthy
Syrian schools	33.68 eyes diseased
Syrian schools	15.00 trachoma
Palestinean schools	39.33 healthy
Palestinean schools	60.67 eyes diseased
Palestinean schools	51.18 trachoma
City schools of Syria	73.30 healthy
City schools of Syria	26.70 eyes diseased
City schools of Syria	9.00 trachoma
City schools of Palestine	57.39 healthy
City schools of Palestine	42.61 eyes diseased
City schools of Palestine	38.00 trachoma
Village schools of Syria	56.48 healthy
Village schools of Syria	43.52 eyes diseased
Village schools of Syria	35.73 trachoma
Village schools of Palestine	26.00 healthy
Village schools of Palestine	74.00 eyes diseased
Village schools of Palestine	64.00 trachoma
Syria girls' schools	9.00 trachoma
Syria mixed schools	12.20 trachoma
Syria boys' schools	19.64 trachoma
Palestine girls' schools	52.19 trachoma
Palestine boys' schools	57.22 trachoma
Palestine mixed schools	60.00 trachoma

A very thorough study of ophthalmia in Palestine was published by T. Harrison Butler in 1907. He limits the use of the term "ophthalmia" to acute and chronic conjunctivitis, excluding trachoma. His studies are based on the abundant material of the British Ophthalmic Hospital in Jerusalem. He found the percentage of trachoma very great, but apparently less than in Egypt. He estimated that

not 10 per cent. of the population of Palestine have absolutely sound eyes. There is no satisfactory explanation for the frequency of ophthalmia in Palestine, when under quite similar conditions of climate, filth, etc., it is not so prevalent in neighboring lands.

In 1903 and 1904 over 2000 cases of leukoma adhærens, staphyloma, and shrunken eyes came to the ophthalmic hospital, forming 15 per cent. of the total new cases during those years. Most were due to summer epidemics, a small minority to trachoma and other diseases. Ophthalmia is always endemic in Palestine. In summer and autumn it becomes severely epidemic.

Bacteriology.—During the period of one year he examined the secretion from 200 cases. The same organisms as are found in Europe and America were likewise present in the more severe oriental ophthalmia. In a certain number of cases, some very acute and complicated with corneal ulcer, organisms could be found in the smears. His results are classified as follows:

SIMPLE OPHTHALMIA—ENDEMIC PERIOD.

(Not Including Cases of Corneal Ulceration.)

	PER CENT.
Morax-Axenfeld diplobacillus	18.3
Koch-Weeks bacillus	18.3
Pneumococcus	18.3
Mixed infection	16.7
Gonococcus	8.4
Sterile	20.0

The mixed infections contained the Morax-Axenfeld in every instance, generally associated with the pneumococcus or a few Koch-Weeks bacilli. In two cases it was associated with the gonococcus. The net result is that during the winter endemic period the majority of the cases contain the diplobacillus of Morax-Axenfeld, followed, in order of frequency, by the pneumococcus, the Koch-Weeks bacillus, and, lastly, the gonococcus. In the endemic period the gonococcus does not always give rise to corneal ulceration. In the epidemic period the gonococcus nearly always causes corneal ulceration, and the ulcers generally perforate.

In very chronic ophthalmias examined during the same period the figures were as follows:

	Per Cent.
Mixed infections	35
Morax-Axenfeld diplobacillus	25
Gonococcus	15
Pneumococcus	5
Staphylococcus	5
Koch-Weeks bacillus	0
Sterile	15

All the mixed infections contained the Morax-Axenfeld diplobacillus, associated with Koch-Weeks bacillus or with the pneumococcus. The Koch-Weeks bacillus was never found alone in a chronic case. The gonococcal cases were all originally acute cases which had become chronic. He regards the Morax-Axenfeld diplococcus as the chief agent in making a simple case chronic.

The percentages during the summer epidemic are very different, as can be seen in the following table, which embodies the results of the examinations of cases of acute ophthalmia:

Acute Ophthalmia—Epidemic Period.

	Per Cent.
Koch-Weeks	66
Pneumococcus	22
Mixed infections	6
Morax-Axenfeld diplobacillus	4
Gonococcus	0
Sterile	2

Severe Ulcers—Examined During the Whole Year.

	Per Cent.
Gonococcus	40
Mixed infections	25
Pneumococcus	15
Koch-Weeks bacillus	10
Morax-Axenfeld diplobacillus	5
Sterile	5

Slight Ulcers—Examined During the Whole Year.

	Per Cent.
Mixed infections	30.4
Pneumococcus	26.1
Koch-Weeks bacillus	21.8
Gonococcus	8.7
Morax-Axenfeld diplobacillus	4.3
Sterile	8.7

ACUTE AND INFLAMED TRACHOMA.

	PER CENT.
Sterile	60
Mixed infections (all containing Morax-Axenfeld)	30
Pneumococcus	10

It will be seen that the variations are very great, and that the gonococcus is responsible for the greatest amount of damage; it occurs in the endemic form without ulceration of the cornea, but not in the epidemic.

Practically none of the sufferers from gonorrheal ophthalmia have gonorrhea. Ophthalmia neonatorum is unknown in Palestine. Butler had never seen a case, nor had Dr. W. E. Cant, who had been in charge of the British Ophthalmic Hospital for some thirteen years.

"Venereal disease is very uncommon in Palestine. Syphilitic iritis and choroiditis, interstitial keratitis, and other similar conditions are very rare in the Jerusalem clinic. Nevertheless, the gonococcus is frequently found, and accounts for many of the lost eyes. I am compelled to come to the conclusion that it is present endemically in the eyes alone, and is passed from eye to eye without the intervention of the urethra."

The epidemic commences annually in July, and rapidly decreases in January. The epidemic is at its height in August, but the virulence increases until November.

During the period of 1900–1905, on which the statistics are based, 34,028 new cases were seen, and of these, 5701 were suffering from ophthalmia, or 16.7 per cent. If we add the 15 per cent. of leukoma, etc., which are the results of ophthalmia, it will be seen that 30 per cent. of all patients who attend an eastern eye clinic suffer from ophthalmia and its results. Epidemics of different years vary greatly in their virulence. There is a certain correspondence between ophthalmia and the mean temperature-curves. The rainfall or humidity does not afford any explanation.

The first sufferers in the epidemic are generally children, often babies one or two years old, and, spreading from the children, the epidemic becomes general.

In spite of the great heat of the summer, the windows and doors are kept shut; the water is scarce, and washing an infrequent luxury. A mother uses her apron to wipe the

eyes of all her children, or, if there be a handkerchief, it is common property. Under such conditions it is manifest that ophthalmia must spread from eye to eye.

The fellaheen, the Arab peasants, lead an entirely outdoor life in the summer, but all the family share a common mattress, and so pus is carried from one person to another.

Flies, he believes, are important agents in the dissemination of ophthalmia. The East abounds with them. Every patient has his own particular swarm, and flies seem to have a special preference for the eye. One often sees a child with several flies settled in and around its eyes, and the fellaheen mothers are too apathetic to brush them away.

Three nurses, under Butler's observation, contracted ophthalmia in Palestine, with the following results: one recovered, one received permanent damage, and the third lost an eye.

Recent Activity in the Study of Ophthalmias and the Campaigns Inaugurated Against Them.—The greatest advance which I observed between my first and second visits to Palestine lay in the efforts that have been made to improve sanitary conditions, and especially those of the eye. There has been great increase in the study of ophthalmic conditions and introduction of prophylactic measures. The physicians of the country, an American society known as the "Hadassah," and the "Jewish Health Bureau" of Jerusalem, founded by Mr. Nathan Straus, have all shown commendable activity in grappling with the problem.

In 1913 a society of physicians in Palestine endeavored to secure definite information by sending out cards of inquiry to all the Jewish schools and kindergartens, with questions as to the condition of the children's eyes, as to whether eye examinations are made and how frequently, as to the care of those affected, etc., and a large number of replies were received. Dr. M. Krinkin, of Jaffa, published the results under the title of "Trachoma in the Jewish Schools of Palestine and the Means of Combatting It."

The report showed that there was the widest difference in attitude toward trachoma, and that there was an absence of any system of prophylaxis, though it must be stated that, in a number of schools, examinations were made before admission and those afflicted with ophthalmias were refused. It was also shown that the number afflicted with ophthalmias was about 21 per cent. in the Jewish schools, against 61 per cent. among the Arabic schools. The fact is emphasized that infection traced to the school is not as frequent as in kindergartens, and, on the other hand, that many children are cured during their attendance at school. He concludes that those forms of trachoma in which there is no secretion may be admitted to school attendance without danger to their comrades, a rule followed in the trachoma provinces of Germany. Owing to the number of young children affected with trachoma he advises that special kindergartens should be provided for these in the larger cities.

In the same year, 1913, Dr. Elias Auerbach, of Haifa, published an article, "Zur Epidemiologie und Bekämpfung des Trachoms in Palästina." He lays stress upon the fact that trachoma is peculiarly a disease of children. He examined 250 trachomatous children under six years of age and found that—

	PER CENT.
59 were affected in first year	23.6
116 were affected in second year	46.4
42 were affected in third year	16.8
18 were affected in fourth year	7.2
12 were affected in fifth year	4.8
3 were affected in sixth year	1.2

In other words, two-thirds were affected in the first two years of life. He is of the opinion that at least two-thirds of all those affected with trachoma are infected in early childhood. He regards trachoma as a family disease, for it is common to find other members of the family affected when one case is discovered.

The danger of infection being proportional to the amount of secretion, it is important, as a prophylactic measure, to reduce the secreting stage into a non-secreting stage, and his experience shows that treatment of a few weeks usually

suffices to accomplish this, and he explains in this way the rarity of infection in those schools in which there is regular treatment.

A systematic campaign against trachoma must have as its chief object the disease among children; the center of work must lie in the school. He regards the complete extinction of the disease as possible in those localities where the population is intelligent. Children who may be the source of infection should be absolutely excluded from school and treated without charge. Cases discovered in school should be followed to their homes and the other members of the family should be examined and treated. In the cities polyclinics should be opened for the treatment of infectious forms of trachoma. Dr. Auerbach closes his article with a call for organization and distribution of the work of sanitation, and for this purpose suggests a trachoma conference which, indeed, was held in the spring of 1914 in Jerusalem.

In 1913 Dr. Arieh Feigenbaum, who has charge of the trachoma division of the Jewish Health Bureau (founded by Nathan Straus), published an interesting report entitled "Die ansteckenden Augenkrankheiten Palästinas und ihre Bekämpfung." His work was begun about June 1, 1913, and his studies were made at the polyclinic and in the schools. In the large majority of cases of so-called "summer inflammation" that he examined the organism found was the Koch-Weeks bacillus. His examinations were mostly made in Jerusalem, but specimens obtained in other parts of Palestine showed the same organism.

The majority of the patients are children, beginning with the new-born, but adults of all ages may be attacked. He often observes the several children of a family, and in the end, one of the parents becoming infected, one after another. In little children both eyes are commonly attacked. In older children it is often limited to one eye.

Though acute conjunctivitis is usually without danger, it probably furnishes very good soil for infection of trachoma in this trachomatous country. His bacteriologic examina-

tions of 126 cases of acute conjunctivitis give the following results:

Koch-Weeks bacillus	83 cases	
Müller's Influenzabacillus (?)	7 cases	
Morax-Axenfeld diplobacillus	4 cases	
Pneumococcus lanceolatus (?)	1 case	
Diplo- (Staphylo-) coccus	2 cases	
Negative	29 cases	= 23 per cent.
	126 cases	

He subsequently found gonococci in one case. The examination of 98 cases of chronic conjunctivitis showed diplobacilli in 44, or 50 per cent.; Koch-Weeks, 4 cases; streptococci, 1 case. In addition, staphylococci and xerosis bacilli were also found. The diplobacilli and Koch-Weeks bacilli were often found together in various stages of trachoma.

In four months he examined 725 dispensary patients, and of these 275, or 39 per cent., had trachoma, but he points out that this percentage is too low, as the examinations of schools, etc., prove. Of these 275 cases of trachoma, 137 were under fourteen years. The material was chiefly made up of native Jews and foreign Jews living for some time in Palestine. His table of all dispensary and institutional cases arranged according to age is as follows:

AGE	NUMBER EXAMINED	SUSPICIOUS	TRACHOMA	PER CENT.
3	41	10	11	26.8
4–7	175	22	52	29.7
8–14	746	37	209	28.0
15–20	368	14	125	33.9
21–30	83	4	21	25.3
31–40	51	5	9	17.6
41–50	32	1	9	28.1
50 and over	29	..	6	20.7
	1525	93	442	28.9

From this table it is evident that a very large majority of patients are infected with trachoma in the early years of life, and that there is a steady increase up to the twentieth year.

The results of his examination of pupils in various schools and inmates of other institutions are given in the following table:*

INSTITUTION	NUMBER EXAMINED	AGE	SUSPECTS	TERMINATED TRACHOMA	MILD TRACHOMA	MEDIUM TRACHOMA	SEVERE TRACHOMA	ENT. SUSP. INC. TERM.	PER CENT.	BLIND IN ONE OR BOTH EYES
Alliance Industrial School	127	7–20	8	5	2	39	4	50	39.4	4
Weavers Industrial School	50	10–69	2	4	..	13	5	22	44.0	2
"Bezalel" Art School	164	10–40	6	9	1	22	1	33	20.1	..
German-Syrian Orphanage	273	6–27	10	14	8	47	14	81	30.0	7
Hebrew Gymnasium	112	6–17	..	1	3	2	..	6	4.5	..
Zelniker Girls' School	121	4–12	4	..	4	22	1	27	22.3	4
Diskin Male Orphanage	151	4–18	6	4	3	25	7	39	25.8	4
3 Jewish Kindergartens of the Hilfsverein	100	3–6	3	..	4	12	..	16	16.0	1
Girls' Lace School	94	11–18	4	5	2	11	2	20	21.3	1
Talmud Torah, "Doresch Zion" Jewish Boys' School	92	6–17	5	3	3	19	..	25	27.3	..
Sephardic Male Orphanage	42	8–17	1	..	3	12	1	16	38.0	2
Persian Talmud Torah (a)	21	5–9	4	..	1	3	..	4	20.0	..
Persian Talmud Torah (b)	32	4–12	3	..	4	9	1	14	44.0	..
Yemenite Talmud Torah (a)	14	10–15	4	..	..	4	..	11	26.0	..
Yemenite Talmud Torah (b)	28	4–11	12	..	..	7	..	..	..	..
Yemenite Talmud Torah (c)	17	3–10	3	..	2	5	..	7	33.0	..
Adult Yemenites	20	25–85	1	1	1	..	1	3	15.0	3
Kurd Talmud Torah	21	4–9	3	..	2	5	..	7	33.0	..

Corneal complications were seen in 23 per cent. of these cases. One hundred eyes showed marked pannus, 13 leukoma, 7 phthisis bulbi, and 5 staphyloma of the cornea. Children up to the fourteenth year showed corneal complications in 9.1 per cent. Those over fourteen showed them in 37.1 per cent.

Only one-half of the cases with corneal complications showed trichiasis. He concludes that trachoma is not a school but a family disease, and that its spread depends on the cleanliness, the hygienic, and the social conditions. The result of the examination of different schools and institutions showed varying figures; the worst conditions were found among the Persian Jews, both adults and children. The Yemenites appear to be free from trachoma when they leave their homes in southern Arabia, but the children, and especially the younger ones, become affected in Palestine.

The causes of blindness are smallpox (5 cases), and espe-

* He uses Hirschberg's classification of trachoma—mild, medium, severe, and terminated.

cially the acute inflammations, certain epidemics of which were exceedingly disastrous. Trachoma, which produces a high degree of visual impairment in many cases, is a much less frequent cause of blindness than are the acute inflammations.

As to the methods of combatting the disease, his suggestions are:

First: Improvement of the social condition of the population, with improved homes and mode of life.

Second: Improvement of the water-supply, especially in Jerusalem, as an essential requisite for cleanliness.

Third: The physicians throughout the country must become interested in the campaign against trachoma; there must be uniform statistics, etc., and the idea of the trachoma conference suggested by Auerbach is approved of.

Fourth: The trachoma campaign should be placed in charge of one central office in Jerusalem, whose duty it should be—

A. To make systematic examination throughout the country according to uniform principles.

B. Uniform guidance so that the necessary decentralization of work will have a common basis.

C. Organization of the campaign in the country by means of free dispensaries, especially in those localities most afflicted. These dispensaries should have a number of beds at their disposal, especially in summer. The ignorant population must be instructed and accustomed to treatment, so as to develop a desire for it. In addition, "movable hospitals," according to Egyptian pattern, would serve for the surgical operations, which shorten the term of treatment.

D. In the cities, the schools, asylums, etc., should be the first to demand attention. School physicians should be secured whenever possible. Children with secretion should not be allowed to attend, but should be treated. Efforts must be made to follow the cases back to their foci of infection in the families, so as to eradicate the infection. The indolent population can best be reached through scattered dispensaries.

E. A central trachoma hospital in Jerusalem, with a sufficient number of beds, should be established, which

would become the central station of all the scattered dispensaries; it should be provided with a dispensary for the free treatment of the poor; opportunity would be offered for scientific investigation and for giving of courses and the opportunity for study to general practitioners.

Feigenbaum gives statistical figures for two colonies, Artuf and Ekron, and he later furnished me with similar figures for Haifa, Rehobeth, Rishon, and Petach Tikvah, of which I give the following:

	Number of Cases	Suspicious Trachoma	Cured	Active	Per Cent.
Colony Artuf	74	9	..	30	40.0
Colony Ekron	207	26	8	71	33.8
Rishon Le Zion:					
Rishon School	111	9	5	11	
Rishon School (Talmud Torah)	47	3	..	12	14.3
Rishon Kindergarten	66	8	..	8	
Rishon Yemenite School	227	9	16	42	18.5
Petach Tikvah:					
Petach Tikvah School	153	..	..	24	15.7
Petach Tikvah School (Talmud Torah)	257	..	..	75	29.2

A valuable article, "Ueber Trachombehandlung in den Schulen," was written by Dr. Ticho, the director of the Jewish Ophthalmic Hospital (Lemaan Zion) of Jerusalem, and was read in April, 1914, at the Trachoma Conference, referred to above. It is based upon the study and treatment of trachoma in a number of schools in Jerusalem, and shows the great variation in the percentage of children affected, according to the character of the population from which the pupils are drawn. The general proportion of children affected was about 28 per cent.; in some it was as low as 15 per cent.; in others as high as 57 per cent.

Among 4525 patients examined, he found 9 cases of pannus, and of these, 8 were girls—2 were only six years old.

There were 11 cases of trichiasis, of which 9 were girls. He points out that the beginnings of the disease are often seen near the external, and more especially the internal, canthus, where granulations are sometimes found in the stage of softening, while the rest of the conjunctiva is still normal. Infection occurs in early childhood, and he has seen one case in an infant but four months old.

In the schools it was in the younger classes (aged five to eight) that the fresh cases were seen most frequently, while in the higher classes cicatricial trachoma was more frequent. Of his 4525 children examined, 1257 were affected with trachoma, which equaled 27.78 per cent. This indicates that the Jewish population is much less affected than the Arabic, among whom trachoma is estimated at 80 per cent. Of his polyclinic cases, 23 per cent. are trachomatous.

From a recent communication from Dr. Ticho I learn that in January, 1915, the number of trachomatous children in the schools under his care (3000 pupils) was reduced from 28 per cent. to 14 per cent. in the course of one year. This shows the effect of proper care and treatment.

The trachoma work of Dr. Ticho and Dr. Feigenbaum in Jerusalem has been helped in no small measure by means furnished from America, but especially by the intelligence and devotion of American graduate nurses sent to Jerusalem and maintained there by the Hadassah Society, a society of Jewish women in America interested in improving the health conditions of Palestine.

Author's Observations.—I reached Palestine on June 22, 1914, and left August 4, 1914. I spent the first and the last weeks in Jerusalem, devoting my time to examining the cases at the Jewish Ophthalmic Hospital, of which Dr. Ticho is Director, but I also visited the British Ophthalmic Hospital, directed by Drs. Herron and Thompson. The material at both was abundant, and there was ample opportunity to observe all forms of trachoma; during my second visit to Jerusalem there were many cases of acute ophthalmia. The acute ophthalmias seen in Jerusalem and elsewhere presented a more angry appearance than those which we commonly

meet; but for their bacteriologic findings, they would often make us suspect gonococcic conjunctivitis. But the latter is evidently rare in Palestine, except during occasional epidemics!

In the company of Dr. Ticho I visited a number of schools, and was able to corroborate his findings and his reports, in particular also his statement that the higher classes present relatively more of the cicatricial stage, while in the lower the earlier stages of trachoma are more prevalent. I also took occasion to visit the trachoma polyclinics which have been opened in the heart of the city by the Jewish Health Bureau.

It was not convenient for me to keep notes of the cases seen in Jerusalem. The observations made in the polyclinics and in the hospital, as well as the examination of the school children, enable me, however, to confirm the statements made by Drs. Ticho and Feigenbaum, referred to above.

SAFED.—The relatively important part played by trachoma among the eye-diseases of a Palestinean clinic will be seen from the following statistics of patients examined by me during the second week of July in Safed, a town of 20,000 inhabitants, situated at an elevation of 838 meters above sea-level, on the foot-hills of the Lebanon Mountains. The total number of patients seen was 584. Of these, there were the following:

Trachoma, stage I	5
Trachoma, stage II	52
Trachoma, stage III	329
Trachoma, suspicious	36
Trachoma, cured	48
Total, 470 (80 per cent.).	
Acute conjunctivitis	2
Simple conjunctivitis	82
Normal conjunctiva	27
Phlyctenular keratitis	2
Blepharitis	3
Pterygium	9
Pterygium, false	2
Chalazion	1
Epithelioma of lid	1

Tuberculous cicatrix of lid	1
Ptosis	2
Strabismus, convergent	24
Strabismus, divergent	5
Detachment of retina	3
Choroiditis	3
High myopia	3
Glaucoma	17
Vitreous opacities	4
Cataract, senile	22
Cataract, complicated	7
Cataract, traumatic	2
Atrophy of optic nerve	1
Traumatic leukoma	1
Traumatic phthisis bulbi	1

The complications of trachoma are shown in the following table:

Shrunken conjunctiva, trichiasis, and entropion	87
Corneal ulcer, opacity, and pannus	107
Keratoconus	2
Leukoma, phthisis bulbi, buphthalmos	39
Dacryocystitis	23
Argyrosis (intense)	8
Calcareous deposits and degeneration of palpebral conjunctiva	17

The number of blind is seen from the following:

Blind in one eye (1 due to smallpox, several due to glaucoma)	14
Blind in both eyes (1 due to smallpox; 1 due to trauma; several due to glaucoma)	21

In both classes most were due to leukoma and phthisis bulbi, following acute conjunctivitis.

Operations performed:

Pterygium	2
Senile cataract	3
Optical iridectomy	1
Trichiasis, entropion, and other lid operations	15

In 62 cases expression of granulations was performed.

In 27 cases the conjunctiva was specially marked "normal." A man aged twenty-five had perfectly normal conjunctiva, though he had lived in this community thirteen years; another, aged twenty-six, was entirely normal though born in this community. Many of the other cases were like these. Many of the normal conjunctiva cases came for other

ocular disease, and some applied in order to assure themselves that they would not be rejected as immigrants to this country. It was interesting to learn how widely the regulations of the United States government against trachoma are known.

I feel quite safe in stating that some of the cases which are noted as normal had passed through mild attacks of trachoma, for there were not a few cases in which the one eye showed distinct evidences of past trachoma, while the other eye showed no such evidence. At the same time it must be stated that there were cases of trachoma in one eye only, the other being normal. Thus two cases are noted as having simple conjunctivitis in one eye and chronic trachoma, third stage, in the other. One case, aged thirty, had left eye normal, right eye trachoma, cured, with dense corneal opacity. Another, aged eighteen, showed the right conjunctiva normal, while the left had trachoma, second stage (and expression was performed). A child, aged ten, was a most interesting case. The left eye showed normal conjunctiva, while the right presented marked trachoma, second stage, with extensive pannus. This cannot but confirm the impression that trachoma cannot be considered as very contagious, but that it requires long and frequent exposure. A number of cases which presented fresh inflammation gave indication, both from their appearance and their history, of being fresh infection in old cases.

The effects of long-continued trachoma in producing calcareous and other degenerative changes of the conjunctiva were seen in many cases. All operators agree that this is a serious complication in cataract operations, and that it is well thoroughly to curet them before performing any intraocular operation.

The evidences of excessive use of nitrate of silver in the treatment of trachoma were seen in the frequent appearance of argyrosis. In some this stain not only involved the whole palpebral conjunctiva, but the entire ocular as well. Such

treatment is, for the most part, carried out by a class of ignorant druggist-oculists, who daily apply the nitrate of silver solutions, receive a small coin for each treatment, and not only do much harm, but also prevent or interfere with proper treatment by competent physicians. Several cases were seen in which the entire cornea was involved in the stain!

A few cases of unusual trouble ought to be mentioned:

A girl, aged thirteen, was seen whose left eye had been enucleated a number of years before and in whose orbit there had been implantation of skin. When the artificial eye was removed, the orbit was found filled with a mass of black hair—a repugnant picture.

Another case of special interest was a young woman who came in a state of great agitation with her handkerchief covered with blood and who told us that this hemorrhage occurred daily from her eye. There were extensive evidences of cicatricial trachoma, but none of ulceration. Observation proved the source of blood to be from the mouth, and I learned on further inquiry that the condition had lasted a long time. It was evidently a case of grave hysteria.

TIBERIAS.—Another city in which I made extensive examinations was Tiberias, a town of 7500 inhabitants, lying 208 meters below sea-level, on the shores of the Lake of Galilee.

Total number of cases examined (during first week of July), 268.

Trachoma, stage I	11	
Trachoma, stage II	36	
Trachoma, stage III	132	
Trachoma, unclassified	28	
Trachoma, cured	24	
	Total, 231	(86 per cent.)
Acute conjunctivitis	2	
Simple conjunctivitis	6	
Normal conjunctiva	28	
Chronic dacryocystitis	1	
Hordeolum	1	
Pterygium	1	
Senile cataract	5	
Glaucoma	1	
Excessive myopia	1	
Leukoma and phthisis (6 were binocular)	9	

The ages of the youngest cases of trachoma found and the conditions are interesting:

Infant, 6 months	Trachoma, stage II
Infant, 8 months	Trachoma, stage II
Infant, 14 months	Trachoma, stage I (severe)
Infant, 18 months	Trachoma, stage II
Infant, 18 months	Trachoma, stage I (severe)

The operations performed in Tiberias:

Trichiasis and entropion	8
Excision of tarsus	2
Excision of lacrimal sac	1
Expression	2
Iridectomy	1

The condition of the pupils at the schools in Tiberias was very bad. The children were crowded together. I saw children with profuse conjunctival discharge in small, overfilled class-rooms. The ocular conditions were correspondingly bad.

TIBERIAS BOYS' SCHOOL (ALLIANCE ISRAELITE UNIVERSELLE).

	Number Examined	Trachoma I	Trachoma II	Trachoma III	Cured	Suspicious	Conjunctivitis, Acute	Normal
Youngest class	23	2	9	9	2	..	1	..
Second class	21	4	1	15	..	..	..	1
Third class	12	3	1	5	2	1	..	..
Fourth class	11	1	2	5	1	1	..	1
Total	67	10	13	34	5	2	1	2

I was informed by the head teacher that the total number of pupils was 97, and that many were absent because of "eye trouble"! Included among the cases of trachoma there was a second case of acute conjunctivitis; 5 cases had marked corneal opacities; 3 cases had marked trichiasis; of the teachers, 2 had trachoma (stage III) and one had normal eyes; the school servant and her son had trachoma (stage

III). It will be seen that 57 out of 67 children examined had active trachoma (85 per cent.), and it may be added that 11 were noted as very severe; only 2 were found quite normal.

TIBERIAS GIRLS' SCHOOL.

	Number Examined	Trachoma I	Trachoma II	Trachoma III	Cured	Suspicious	Normal
Youngest class	24	6	7	10	..	..	1
Second class	25	9	3	10	3	..	..
Third class	13	3	2	5	1	..	2
Fourth class	30	6	1	13	5	2	3
Fifth class	12	2	2	5	2	..	1
Sewing class	14	..	2	11	1	..	..
Total	118	26	17	54	12	2	7

In this school there were also many pupils absent because of "eye trouble." Of the 118 pupils examined, 97 presented active trachoma (83.6 per cent.). It is interesting to note that one of those included under "normal" was born in Tiberias and was fourteen years of age. Corneal leukomata were noted in 4 cases, entropion in 4 (the youngest being eight years old), and corneal opacities in 2.

JEWISH SETTLEMENTS.—The examination of the newer Jewish agricultural settlements near Tiberias was very interesting, more particularly because they had recently come under the care of an able oculist, Dr. David, of Yemma. I was much interested in his reports concerning a method of prophylaxis that he had introduced two years previously. It consisted in the instillation, once daily, of a solution of 1 per cent. of zinc sulphate and 1:10,000 of adrenalin, in all healthy eyes, adult and children. As a result, I was assured by Dr. David and by the head masters of the schools that no fresh cases of trachoma had developed and that not a single case of acute conjunctivitis had occurred among those treated during the two years it had been used.

YEMMA is a colony near the Lake of Galilee.

	SCHOOL	ADULTS
Trachoma	33	..
Suspicious	1	..
Cured	9	3
Conjunctivitis:		
Simple	6	2
Acute	5	2
Normal	26	5
Total number examined	80	12

Among the school children I found:

2 cases of trichiasis.
4 cases of corneal opacities.
Trachoma, 40 per cent.

MESCHA, at foot of Mount Tabor:

	SCHOOL CHILDREN AGES			
	3–5	6–8	9–11	12–14
Trachoma I	2	2	5	4
Trachoma II	3	2	6	2
Trachoma III	..	1	1	8
Suspicious	3	1	1	..
Normal (including cured)	14	13	15	6
Number examined	22	19	28	20

Percentage of trachoma in all classes, 40.

Note.—Fifteen adults were examined and but one case of trachoma found.

MILHAMIE, a colony situated at the point where the Lake of Galilee flows into the Jordan:

	SCHOOL	ADULTS
Trachoma (all forms)	8	1
Cured	19	6
Conjunctivitis (simple)	5	4
Normal	9	6
Total number examined	41	17

Among the children there was:

1 case of corneal opacities.
1 case of leukoma.
Trachoma, 20 per cent.

Thirteen cases presented very marked argyrosis, the effects of the work of a druggist before Dr. David's arrival.

Among the adults I found:

Trichiasis	1
Corneal opacities	2
Degenerative changes in conjunctiva	2
Cataract	1
Quinin blindness (in a girl of sixteen, acquired at the age of four years)	1

SEDGERA, a colony near Mount Tabor:

	CHILDREN	ADULTS
Trachoma (unclassified)	7	..
Trachoma I	5	..
Trachoma II	13	..
Trachoma III	4	2
Trachoma suspect	4	..
Cured	11	1
Conjunctivitis (acute)	1	..
Normal	19	4
Total number examined	64	7

The percentage of trachoma among the children is 45. Pannus was noted once (in a lad of seventeen years). Leukoma was noted once (in an adult).

In a severe case of trachoma, stage II, in a boy of five years I learned that the condition had been one of mild trachoma, stage I, when an acute conjunctivitis was contracted, and after this had passed off the trachoma was found much aggravated.

I noted here, as elsewhere, a large number of cases of trachoma cured in which there is no evidence of past trachoma or only very slight. Pannus was not seen in any of these cases.

PORIAH, a new colony overlooking the Lake of Galilee:

	ADULTS AND CHILDREN
Trachoma (unclassified)	1
Trachoma I	11
Trachoma II	4
Trachoma III	16
Suspicious	6
Cured	9
Conjunctivitis (simple)	6
Normal	14
Total number examined	67

The percentage of trachoma is 50. Seven cases were marked as "severe." Corneal opacities were noted once. Phthisis bulbi was noted once. Trichiasis was noted once.

BETHANIA and DAGANIA, situated near MILHAMIE:

	ADULTS
Trachoma	6
Conjunctivitis (simple)	6
Conjunctivitis (acute)	2
Normal	9
Total number examined	23

Inasmuch as these figures are small, I have added the number of adults of Milhamie, Bethania, Dagania, and Yemma, which total 52, and find 15 cases of trachoma, or a little less than 30 per cent.

HAIFA.—A city of 16,000 inhabitants, situated on the Mediterranean, at the foot of Mt. Carmel. The figures for Haifa were given me by Dr. Feigenbaum, and were prepared by Dr. E. Auerbach in June, 1914. I visited the schools with Dr. Auerbach shortly afterward, and examined the cases of trachoma and corroborate his figures:

	NUMBER OF PUPILS	TRACHOMA	CURED	SUSPICIOUS	PER CENT.
Boys' school	167	54	12	9	39.5
Girls' school	157	40	12	10	33.1
Hebrew Realschule	100	10	3	2	13.0
Kindergarten	35	3	..	3	8.5

This well illustrates the difference between schools which are under the care of an intelligent physician and those in which there is no such systematic care. The "Realschule," a modern institution in all respects, is under Dr. Auerbach's supervision, and no pupils affected with trachoma with any secretion are permitted to attend. The same rigid rule applies to the kindergarten, where there were formerly 38 per cent. trachomatous. The entire school is reëxamined every three months, all new applicants are carefully examined, and none showing secretion are admitted. The cases of trachoma which are allowed to attend are separated from other healthy children and are treated daily.

The following settlements which were visited are in the south, near Jaffa. They are older settlements; it is only recently that systematic treatment has been undertaken.

REHOBETH, a town of 1500, established twenty-five years ago, a few miles south of Jaffa, is a rather successful settlement. I spent a number of days in Rehobeth, and examined the eyes of a considerable number of the inhabitants in the "dispensary," with the kind assistance of the intelligent physician of the colony, Dr. Muskovitz. The total number examined was 72.

Trachoma in various stages	20
Trachoma cured	16
Simple conjunctivitis	17
Acute conjunctivitis	1
Trichiasis	2
Corneal opacities and pannus	8
Leukoma (including phthisis bulbi)	4
Lacrimal disease	1
Phlyctenular keratitis	1
Blepharitis	1
Cataract	2
Glaucoma	1
Strabismus	1
Blind in one eye	3
Blind in both eyes	1

Active trachoma, 27.7 per cent., and if cured cases are added, 50 per cent.

	Number Examined	Trachoma	Cured	Conjunctivitis		Normal
				Simple	Acute	
Kindergarten	33	9	7	2	..	15
School, lowest class	19	6	1	4	..	8
School, second class	20	3	5	1	..	11
School, third class	25	10*	3	1	..	11
School children examined at dispensary	8	3	1	..	1	3
Talmud Torah School	22	9	5	4	..	4
Yemenite School	22†	16	..	1	4	1

* Including one case of marked trachoma in left eye; right eye normal.

† Includes one case of leukoma (in a child of five years) from gonorrheal ophthalmia one year ago. There were two cases, the other an adult. In both bacteriologic examination confirmed the diagnosis. The trachoma conditions in this school were very bad.

The percentage in the different schools, accordingly, varied from 27 to 73! The average was 31.5 per cent.

EKRON, a settlement near Rehobeth, which for years has been noted for severe trachoma, gave the following in 152 cases examined:

Trachoma in various stages	63
Trachoma cured	43
Simple conjunctivitis	7
Acute conjunctivitis (pseudodiphtheric)	1
Normal conjunctiva	38
Total number of cases examined	152

The percentage of trachoma among those examined was 41, and if the cases of past trachoma are added, it is 69 per cent. The number of cases of *severe* trachoma was unusually large, and is shown by the following complications:

Trichiasis and entropion noted in	5
Corneal opacities, including pannus, in	12
Leukoma	7
Phthisis bulbi	5
Staphyloma of cornea	1
Blind in one eye	3
Blind in both eyes	7

Among the other conditions were:

Strabismus	6
Pterygium	1
Glaucoma	5
Phlyctenular conjunctivitis	3
Blepharitis	1

Of the total number of 154 cases, 109 were under sixteen. Of these, there were:

Trachoma in various stages	47
Trachoma cured	31
Simple conjunctivitis	6
Acute conjunctivitis	1
Normal conjunctiva	24

The percentage of trachoma among the children was, therefore, 43, and if the cured cases are added, 71.

Careful inquiry was made through the attending physician, who kept a careful record of each family and member of the community, concerning all the members of the families in which any children were examined. Definite information could be obtained concerning a total number of 200, and of these, 123, or 61.5 per cent., had active or cured trachoma.

A few families were of special interest:

Family A:

Father, age forty-three, trachoma cured.
Mother,
Daughter, age twenty, trachoma, stage II–III.
Daughter, age nineteen, trachoma, cured.
Daughter, age sixteen, trachoma, ?
Daughter, age fifteen, trachoma, almost cured.
Son, age fourteen, trachoma, cured.
Son, age ten, trachoma, cured.
Daughter, age nine, trachoma, almost cured.
Son, age six, trachoma, almost cured.
Son, age four, trachoma, almost cured.
Son, age three, trachoma, almost cured.
Son, age one, normal.

Besides the above, there were numerous cases in collateral branches of the same family. The physician pointed out that there were a number of families who lived under particularly bad hygienic conditions, and who were the chief sufferers in the colony. Thus family R, consisting of the parents and eight children, were all affected without exception.

A case to which I desire to direct special attention as bearing on reinfection was a boy, aged ten, who had been completely cured, and after some time was reinfected, and at the time of examination presented trachoma, stage II, in severe form.

In Ekron I performed a large number of expression and curetment operations.

The opportunity to examine Bedouins and Fellaheen is not easily obtained. I saw numerous Arabs in the city clinics, but they were for the most part city dwellers. I was, therefore, glad of the chance to make the following observation:

On the road to Sedgera from Yemma I saw a colony of Bedouins (not true nomads, but those remaining around the same locality and living in huts). I examined ten children, about two to five years of age, and several infants nursing at the breast, as well as older girls and women—all had trachoma except two, who were apparently normal; one or two young children showed fairly good conditions with evidences of scar tissue in the tarsal cartilage. The older girls and women all had marked trachoma, stage III. The young infants showed marked trachoma with much secretion—one infant was only five months old.

The Arabs of this class are quite generally trachomatous, according to the information I gathered from physicians familiar with them. The forms which they present in adult life are the very worst. Certainly the most aggravated conditions seen in the clinics were among Arabs.

The total number of examinations made by me (exclusive of those in Jerusalem) exceeded 2000 cases. One-half of these were school children. Among these I found 54 per cent. presenting active or terminated trachoma.

SUMMARY.

The conclusions to be drawn concerning the conjunctival inflammations of Palestine may be briefly stated as follows:

Simple conjunctivitis is of frequent occurrence and is easily accounted for by the combined irritation of the bright sunlight and the dust.

Vernal conjunctivitis is not common, but isolated cases are seen in different parts of the country, usually associated with trachoma. Both of these diseases naturally tend to spread trachoma and acute conjunctivitis; the irritation, which induces rubbing the eyes with contaminated fingers, readily furnishes the opportunity for infection. It is impossible to state whether an already inflamed conjunctiva is more susceptible to trachomatous or other infection, though this may

be regarded as probable. The common manner of carrying the contagion is certainly direct—by hands, clothes, dirty washing utensils, and flies.

The rarity of phlyctenular ophthalmia impressed itself upon me, as it has upon other observers. This is not easily explained, for malnutrition is exceedingly common. The out-of-door life may be the reason for the rare occurrence.

Chronic angular conjunctivitis (Morax-Axenfeld) is of frequent occurrence, commonly associated with chronic trachoma.

Acute conjunctivitis: the description of these cases may best be given in the words of Butler:

> The onset is rapid, one eye being generally affected first. Cases may commence as catarrhal conjunctivitis, which may get well, or a mucopurulent discharge may appear and may eventually become purulent. Some cases have from the first a mucopurulent discharge.
>
> The mild cases show a pink injection of the conjunctiva. In Koch-Weeks infection the slight cases may have small or large subconjunctival ecchymosis, whereas in pneumococcal infection there is often chemosis, even in mild cases. . . . The cases in the summer epidemics are so severe that one cannot form much from the clinical picture as to the nature of the infection. . . . In children the conjunctiva becomes swollen and sponge-like, and it bleeds at the slightest touch. It may give rise to an enormous ectropion, which can be reduced only with difficulty and at once recurs.
>
> Under treatment ophthalmia lasts from a few days to a month or more, but untreated the cases become chronic and drag on for months.
>
> The typical epidemic case caused by the Koch-Weeks bacillus is generally easily cured; pneumococcal cases are more liable to become chronic.
>
> The Koch-Weeks type has a great tendency to relapse, and there is no immunity whatever. One individual may have three or four relapses in a summer.
>
> Prognosis is generally good in Koch-Weeks infection if the eyes come under treatment at once. The same is true in a

slightly less degree of the pneumococcal infection, but there is much greater danger to the cornea. . . . In gonococcal infection the prognosis is always bad, and generally very bad.

Of the 5700 cases of ophthalmia collected by Butler, 22 per cent. developed corneal lesions, and 46 per cent. of these perforated. "Of these perforations, at least 50 per cent. were due to the gonococcus, either alone or in mixed infection."

One is impressed by the fact that the types of acute ophthalmia met with in Palestine are markedly more severe than those which we commonly see, and that they more frequently lead to corneal involvement and destruction. This is probably accounted for by the prevalent malnutrition, the wide-spread ignorance, and the poor care of the patients. One is likewise impressed by the fact that the epidemics of certain years were uncommonly destructive to the cornea. A large number of the cases of leukoma are found to have resulted from a few such malignant epidemics.

The relative frequency of the various organisms studied a number of years apart by Butler and by Feigenbaum show that the Koch-Weeks bacillus is the most frequently found in acute epidemic ophthalmia. Both observers noted it in 66 per cent. of the cases.* Their findings as regards the frequency of other organisms do not agree, though both find the pneumococcus, Morax-Axenfeld diplobacillus (and Feigenbaum, the influenza bacillus), and a varying proportion of negative results.

The gonococcus was not found by either in the acute epidemics which they studied. I am of the opinion that the gonococcus is the probable cause of the rare and extraordinarily malignant epidemics. I cannot close this statement without again referring to the absence of gonococcic ophthal-

* Meyerhof found almost the same percentage in Egypt (*Klin. Jahresbericht,* 1911).

mia neonatorum, which Butler emphasizes, and which my own inquiries corroborated.

It is difficult to give definite figures concerning the frequency of blindness in Palestine. No comprehensive numbers have as yet been collected. Feigenbaum found 43 cases of monocular and binocular blindness among 1760 cases examined; of these, 28 cases were found among 1479 school children (2 per cent.). This is lower than the figure given by MacCallan for the Egyptian schools (3.3 per cent.). My own figures are even somewhat less than those given by Feigenbaum.

Trachoma.—It is difficult to state accurately the frequency of trachoma in Palestine. It certainly does not rise to the large percentages which are recorded for Egypt by MacCallan, as being from 90 per cent. to 95 per cent. in the schools. At the same time, in some localities and among certain classes of the population the percentages may approach those of Egypt. There are marked differences between different classes, the frequency being in direct ratio to the unhygienic conditions under which they live. Germann found the frequency of trachoma in the Palestinean schools to vary from 51 per cent. to 64 per cent. Ticho's figures for the schools of Jerusalem before systematic treatment was introduced showed a variation between 15 per cent. and 57 per cent., with a general average of 28 per cent. Feigenbaum's highest percentage for schools and orphan asylums was 44 per cent. My own figures for schools outside of Jerusalem vary from 8 per cent. to 85 per cent., with an average of 54 per cent.

The worst conditions that I found were in the schools of Tiberias, where there were very few healthy eyes.

In most schools, and even among the class of patients that apply for treatment at an ordinary clinic, there is a considerable number of natives who show no evidences of past or present trachoma. Thus in Safed 20 per cent., and in

Tiberias 14 per cent., of these patients were free from evidences of trachoma.

A most interesting fact which strikes the observer familiar with trachoma as it appears in America and Europe is its occurrence in Palestine in early childhood. It is estimated that two-thirds of all cases originate in early childhood (Auerbach). The youngest infants which I saw with well-marked trachoma were five and six months of age.

In children the very large granulations of the second stage, covering the entire tarsus, with much secretion, are characteristic. While for purposes of classification the three stages of Raehlmann are generally adopted, it is often found that the first and second stages, or the second and third stages, and sometimes all three, are seen in the same conjunctiva.

Trachoma in the young presents other peculiarities, principally the relative absence of corneal, lid, and lacrimal complications. This fact is pointed out by all observers. Feigenbaum finds that 9 per cent. of cases of trachoma up until the fourteenth year show corneal involvement, against 37.7 per cent. in adults. Ticho found pannus and trichiasis much more rarely still in children, and my own observations accord with those of Ticho.*

Another characteristic of the trachoma of children is its

* My observations are in accord with those of Germann, Feigenbaum, Ticho, and others in respect to the infrequency of complications in trachoma among children. The total number of children examined outside of Jerusalem was upward of 1000. Among these I saw pannus in but 5 cases under thirteen years of age. The youngest was a child, aged five, with severe trachoma, Stage III, and pannus in both eyes. The next older was an idiotic girl, aged nine, with severe trachoma, Stage II–III, and pannus. The next a child of ten with normal left eye and trachoma, Stage II, marked in the right eye, with pannus and entropion. The last is a child of twelve with trachoma, Stage III, cured in the left eye and active in the right, with pannus.

Besides the case of entropion mentioned, this complication with trichiasis was observed in three children. The youngest was eight, the others were eleven and twelve respectively. Other complications, such as corneal opacities, were observed more frequently. Lacrimal disease was noted in a child of twelve. Marked shrinking of the conjunctiva was observed rarely under fourteen years of age, and noted in but one case, a child of eleven. Leukomata were seen more frequently, but were generally found to have resulted from acute "summer inflammations."

readier response to treatment, especially to surgical treatment, and its definite tendency toward spontaneous cure in many cases. One is struck, in examining large numbers of persons, by the frequency with which cases of cured trachoma are met with. In many of these there are only the faintest cicatrices of trachoma. This emphasizes the advisability and the necessity of early surgical treatment and the careful after-treatment in children. I refer to curetage and the various methods of expression.

Concerning the danger of infection in children, it is probable, and most of those familiar with the conditions in Palestine have concluded, that infection is chiefly spread in the home. It is likewise spread among children at play, and in those schools where they are huddled together and where no precautions whatever are taken. On the other hand, I was assured by a number of competent observers that infection seldom, if ever, occurs in schools in which the simplest precautionary measures are taken.

I may mention parenthetically that all agree that acute conjunctivitis and trachoma do not react favorably upon each other. All evidence goes to show that acute conjunctivitis often serves as the period of infection for trachoma, and, what is still more certain, that acute conjunctivitis engrafted upon trachoma leaves it an aggravated condition.

Concerning trachoma in the old, it is necessary to mention the frequency of marked degenerative changes in the conjunctiva, resulting in minute cystoid collections containing colloid, calcareous, or purulent matter, a condition well described in MacCallan's monograph. These are a source of danger in intraocular operations and require thorough and previous curetment.

Another peculiarity of trachoma in the aged consists in the development of a marked thickening of the conjunctiva, without the presence of granulations, but with chronic discharge and irritation.

That there are differences in susceptibility to the infection of trachoma and that there are those who present relative immunity must be assumed. Otherwise it is difficult to explain those cases which, in spite of constant exposure to infection throughout their lives, have escaped. It must be borne in mind also that cases of unilateral trachoma are occasionally seen.

In conclusion I must express my hopefulness concerning the trachoma situation in Palestine. The prophylactic agencies referred to above have already resulted in distinct improvement of the conditions in many schools. With improvement in social and economic conditions, with hygienic reforms, with better knowledge and understanding on the part of the population of the effectiveness of prophylactic and curative treatment, we may look for great reduction in the frequency of trachoma in Palestine.

REFERENCES.

Auerbach: Zur Epidemiologie und Bekämpfung des Trachoms in Palästina, *Deutsch. med. Wochenschrift*, 1913, No. 37.

Butler: The Clinical Features, Bacteriology, and Treatment of Acute Ophthalmia in the East, with an Analysis of 5,700 Cases. Royal London Ophthalmic Hospital Report, 1907.

Feigenbaum: Die ansteckenden Augenkrankheiten Palästinas und ihre Bekämpfung, Jerusalem, 1914.

Germann: Augenaerztliche Beobachtungen in Syrien und Palästina; speciell über das Trachom in diesen Ländern, *Centralbl. f. Augenheilkunde*, 1896, p. 386.

Hirschberg: Aegypten, Leipzig, 1890.

Howe: On the Influence of Flies and the Spread of Egyptian Ophthalmia, Seventh International Ophthalmic Congress, Heidelberg, 1908, p. 283.

Krinkin: Trachoma in the Jewish Schools of Palestine and the Means of Combatting It, Jaffa, 1913. (Hebrew.)

MacCallen: Trachoma and its Complications in Egypt, University Press. Cambridge, 1913.

Meyerhof: *Klin. Monatsbl. f. Augenheilk.*, 1905, 1908, 1910; *Ann. d'Ocul.*, 1906, 1909; *Rev. Gen. d'Opht.*, 1909; Über die ansteckenden Augenleiden Ägyptens, ihre Geschichte, Verbreitung, und Bekämpfung, Cairo, 1909, etc.

Meyerhof: *Klinische Monatsblätter für Augenheilkunde*, June, 1912, p. 641.

Ticho: Über Trachombehandlung in den Schulen, *Zeitschrift f. Augenheilk.*, 1914, vol. xxxii, p. 368.

DISCUSSION.

DR. LUCIEN HOWE, Buffalo, N. Y.: The fact to which I venture to call attention is one which Dr. Friedenwald has probably observed. It is the effect of nutrition in the class of cases to which he refers. That is, to my mind, a very important factor. The food of the poor in Egypt is largely starchy, and the people live under conditions in which the nutrition suffers. This indicates again the importance of our taking into account good diet and good hygiene in the treatment of such cases.

DR. G. O. RING, Philadelphia: I should like to ask Dr. Friedenwald whether he can tell the extent to which grattage is practised there as a curative method for these conditions, and whether the method that I have found useful following grattage, the application of trichloracetic acid in 3 or 4 per cent. solution, is or is not valuable.

DR. JOHN E. WEEKS, New York: I am sorry that I did not hear the entire paper of Dr. Friedenwald. I understood him to say that three stages of trachoma were observed from time to time in the same eye. In a somewhat extensive study of this disease I have never observed this condition. The early stage of trachoma, as we all recognize, is the stage of hypertrophy, in which the granules first appear. In the first few days the granules or follicles may be hidden in thickened conjunctiva, becoming visible later. In the second stage, that of coalescence, these granules coalesce more or less completely. Then comes the stage of atrophy. To my mind, it is an impossibility for the three conditions to exist at once.

Another statement made was that acute conjunctivitis does not have a beneficial effect on trachoma. That is also a statement at variance with the experience of most of us, I think. The induction of acute conjunctivitis has been brought about by various methods in cases of trachoma, such as the use of jequirity and the infection of the eye with gonorrheal pus, and I have myself observed many cases of trachoma in which acute contagious conjunctivitis was engrafted. We all know that in many cases the effect has been an improvement in the condition of the trachoma after

the acute process has subsided. I have repeatedly observed this after acute contagious conjunctivitis. In 1909, at Budapest, Goldzieher presented a case in which he had inoculated the eyes with the gonococcus, and the trachoma had disappeared. Of course, this practice with us is obsolete, but I wished to call attention to the fact that at that late date Goldzieher had presented such a case. Although there may be exceptional cases in which the trachoma is not benefited by the induction of an acute process, we find, as a rule, that it is benefited when this takes place.

So far as the transition of acute contagious conjunctivitis into trachoma is concerned, this, I think, never occurs except through added infection. That the conjunctiva may be more susceptible to the infection of trachoma during and immediately after acute contagious conjunctivitis is a view with which I fully agree.

DR. HARRY FRIEDENWALD, Baltimore, Md.: Dr. Howe's explanation for the rather remarkable manner in which acute conjunctivitis in the Orient causes destruction of the cornea, namely, the prevalent malnutrition, was the same to which I had arrived.

In answer to Dr. Ring I would say that the modern physicians in Palestine are using the various surgical methods of treatment of trachoma, grattage, roller forceps, expression, etc., applying afterward some mercurial or copper solutions.

Dr. Weeks is perfectly correct in his classification of the stages of trachoma: first, the follicular; later on, the large granular; and subsequently, the cicatricial form. When I stated that we often have two stages combined, I meant that different parts of the conjunctiva may show two different stages. We may have the follicles distinct in one part, while in another part large granules are formed. Sometimes we see the cicatrices beginning in still another part of the conjunctiva. The explanation for this is the exceedingly chronic course of these cases, which have gone untreated for a long time, and sometimes perhaps reinfection with fresh trachoma in an older case in which the cicatricial stage had already set in.

I went to Palestine with the same prejudice concerning the

good effect that acute conjunctivitis is said to have upon trachoma, but I saw there a number of cases in which mild trachomas were very greatly aggravated after acute ophthalmias. I did not see any case that had passed through a gonococcal ophthalmia; but those with milder trachoma certainly were aggravated in a number of instances by the acute attack of ophthalmia.

PEMPHIGUS OF THE CONJUNCTIVA.—REPORT OF TWO CASES.

EDWARD STIEREN, M.D.,
Pittsburgh, Pa.

Pemphigus of the conjunctiva is a rare condition, occurring 5 times in 45,000 ophthalmic patients, according to Franke,[18] 2 times in 22,000 ophthalmic cases, according to Pergens.[28]

Its occurrence is almost invariably incident to a general bullous eruption of the body. We may find it accompanying epidermolysis bulla, bullous dermatitis, pemphigus vulgaris, acute pemphigus, and pemphigus neonatorum syphiliticus.

The frequency of ocular involvement in pemphigus of the skin is apparently not great, since Hebra did not observe it once in 200 cases.[27] The stage of vesication is rarely observed in the eyes, since the epithelium of the conjunctiva is so much more delicate than that of the skin that the vesicles soon burst, and the ocular disease appears as denuded areas or recent cicatrices, soon followed by great contraction, which gives the eye a scalded and lifeless appearance. According to Bordley,[6] the conjunctival lesions may appear as petechiæ, becoming confluent in the ocular conjunctiva, but remaining discrete in the lids.

I have observed two cases of pemphigus of the conjunctiva in approximately 20,000 ophthalmic patients.

CASE I.—Referred by Dr. W. J. Stewart, August 1, 1907. J. L. S., white, farmer, aged forty-seven years. Eyes became affected a month or six weeks before, during an attack of dermatitis bullosa of his entire body. The attending physician stated that the odor from the patient in this condition was overpowering.

Ocular Examination.—Intense photophobia. Moderate entropion of both lower lids. Conjunctiva glistening and shrunken, with slight discharge of mucopurulent character, which contained only the usual conjunctival flora. A symblepharon between the lower lid and eyeball, extending from the inner canthus and occupying about one-third of the sulcus in the left eye, and several ribbon-like, similar adhesions in the right eye.

Both corneæ diffusely cloudy, the left being encroached upon by what appears to be an internal pterygium, minus the usual blood-vessels. The patient suffered intensely from corneal irritation, which could, however, be relieved with holocain. No staining of the cornea with fluorescein, but several small areas of the conjunctiva in each eye were denuded.

The patient, a syphilitic, had been taking enormous doses of potassium iodid. To this was added mercury bichlorid, $\frac{1}{8}$ grain, three times daily. Locally, 20 per cent. argyrol every three hours, 10 per cent. dionin, and 1 per cent. atropin three times daily were employed for two weeks. The photophobia gradually improved under this treatment, and it became possible to estimate his vision at the end of two weeks, which was approximately $\frac{6}{80}$ with either eye.

September 5th he had perfect control of the eyes in sunlight with smoked glasses on and without glasses indoors. Vision had improved to $\frac{6}{36}$ with the right eye and $\frac{6}{20}$ with the left, the corneal haze having cleared up to this extent. A canthoplasty in each eye, with dissection and turning-in of the symblepharons, resulted in satisfactory amelioration of the entropion.

September 14th atropin was discontinued, and pilocarpin in a weak solution substituted. Mercury bichlorid had steadily been exhibited in increasing doses, the point of tolerance being $\frac{1}{2}$ grain three times daily. The use of dionin was also persisted in.

October 30th the patient returned to his home in the coun-

try, with instructions to continue both local and internal treatment. V. R. E. = $\frac{6}{36}+$; L. E. = $\frac{6}{20}+$. Subsequently he suffered a violent return of his previous skin disease, and in a despondent mood committed suicide by shooting.

CASE II.—Charles S., white, aged six years. Admitted to the South Side Hospital February 10, 1915, suffering with a number of skin lesions over almost the entire surface of the body, including the scalp and face, and inability to open his eyes.

His parents supplied the following history: General health is good; appetite poor; bowels regular. Has had none of the acute exanthemata, but suffered an attack of pneumonia in infancy. Child was vaccinated about September 15, 1914. About four weeks later he developed a large bleb on the dorsal side of the left forearm, followed by a similar lesion on the posterior surface of the left ear; then numerous blebs appeared on the arm, legs, and trunk. These varied in size from a pin-head to a pea, and would rapidly coalesce, forming at times blebs as large as a walnut, which would rupture spontaneously, liberating a large quantity of serum, and followed by raw, bleeding surfaces which would take on thick incrustations.

Similar lesions occurred on the eyelids and in the anterior nares. The child persisted in picking off the crusts on the eyes, which refreshened the lid margins and resulted in the lids growing together.

Upon admission: Child is irritable, underweight, and anemic. Large areas of the body are covered with lesions varying in size from a dime to a dollar, covered with red incrustations. No bullæ in evidence.

The eyelids, especially near the margins, are covered with tenacious incrustations, which leave bleeding surfaces when removed. There is marked entropion of both upper and lower lids. The lids cannot be separated, being united by a continuous band of adhesion occupying the outer half of the left lids and the inner half of the right lids.

Under ether the adhesions between lids were divided and an external canthoplasty was performed in each eye. Numerous symblepharons had formed between the lids, both

upper and lower, of both eyes. These were dissected up and the defects covered, the conjunctiva being so friable and shrunken that it had to be handled with the greatest care.

The right cornea was found to have a general diffuse superficial haziness, while deep in the central corneal stroma was a yellow macule about 3 x 4 mm. in diameter. The left cornea had the same superficial haze as the right, but the stroma was clear.

The after-management of the child was fraught with the greatest difficulties on account of the blepharospasm, which was so intense that all endeavors to cleanse and treat the eyes would result in the integument of the upper lids breaking down (literally peeling off) when forcible opening with the fingers was attempted, to be followed by granulating surfaces which bled profusely whenever disturbed.

Finally, after two months of treatment, the raw lesions about the lids became covered with epithelium and the external canthal wounds healed. The conjunctiva now was pale, glistening, and shrunken, and the left cornea had developed the same yellow macular degeneration first noted in the right eye. The child can at times overcome his blepharospasm, and will open his eyes momentarily, but it is evident that he is quite blind.

A number of bullæ appeared on his body, mainly on the lower arms and legs, during his stay in the hospital. The serum from these bullæ was repeatedly examined, and cultures made in the laboratory, the organisms present being diplococci, slightly larger than gonococci. There was an occasional rise in temperature to 100°, which was invariably followed by the appearance of fresh bullæ.

Repeated examinations of the urine were negative, as were also Noguchi and Wassermann tests. In spite of the latter the patient received mercury, in the form of gray powder, and Fowler's solution persistently for two months.

Etiology.—Pemphigus of the skin in any of its forms may accompany or follow many conditions, but there does not appear to be any particular predisposing factor. One of my cases occurred in a syphilitic. This has been observed also by von Marenholtz [35] and by Fehr.[17]

In my other case the disease appeared four weeks after vaccination. In regard to this factor Bowen,[10] who in 1901 reported six cases of bullous dermatitis following vaccination in children, states: "I have emphasized the fact that I cannot prove that they were caused or influenced by the vaccination." Howe,[21] in 1903, reported 10 cases of bullous dermatitis in adults following vaccination, 6 of whom died. Because of this high mortality he is of the opinion that the disease was the result of infectious material introduced at or after vaccination. Pernet and Bullock[29] report 17 cases of an acute bullous eruption, the infection, probably a specific microörganism, taking place through a wound. Eight of these cases occurred in butchers, and the mouth, palate, eyes, and nostrils were always affected. In eight of their other cases the occupation of the patients brought them in contact with animals.

These observers call attention to the fact that bullous eruptions occur not infrequently in horses, cattle, dogs, and pigs. Ballard[2] reports a case of pemphigus of the skin, mouth, and throat of a young man who contracted the disease through an open wound on the dorsum of the hand while milking cows who had bullæ on their udders. Corlett[14] reports a case of erythema circinatum bullosum et hæmorrhagicum following a gunshot wound and terminating fatally from streptococcic infection.

Most of the reporters remark on the intense fetid odor. (This was present in my first case, but not in the second.)

Bacteriology.—Xerosis bacilli in the latter stages of pemphigus of the conjunctiva are usually present in large numbers. It is not probable that they have anything to do with the condition, as they are found frequently in the conjunctival sac in health, and simply find in the degenerated epithelium and in the denuded areas favorable soil for growth.

In my first case examination of the discharge from the eyes showed nothing significant. In the second case serum

from freshly formed bullæ on the skin repeatedly revealed the presence of diplococci about one-third larger than gonococci, staining by Gram's method, and apparently the same as those found by Demme,[16] Dahnhardt,[15] and Bleibtrau.[5] The organism they describe is a diplococcus, much resembling the gonococcus, but larger, its two opposing surfaces being flattened. It grows well on agar and serum, forming round white colonies, and stains well with all anilin dyes and by Gram. Other observers have found streptococci and staphylococci, but these, when found, are usually regarded as accidental infections.

Pathology.—A comparatively recent and excellent histologic study of pemphigus of the conjunctiva is that of Adam,[1] who finds that the changes are decidedly different than those found in pemphigus of the skin. The difference consists mainly in the specimens examined by him, in the absence of bullæ; an inflammation of the subepithelial and adenoid elements, later passing into cicatrization, with raised nodules on the surface of the conjunctiva. The shrinking of the conjunctiva he finds is not due to plastic adhesions of ruptured vesicles in the supporting basal membrane, but to cicatricial changes in each nest of infiltration. The bulbar conjunctiva soon becomes tightly adherent to the sclera at the site where the nodules occur, with a shrinking of the same, while the palpebral conjunctiva retains more or less of its laxness. This results in the formation of vertical folds in the conjunctiva of the culdesac, which look like adhesions or symblepharons, and which can be mistaken for such.

Adam found in his corneal sections that the process consisted of a denudation of the epithelium, followed by an irregular regeneration of exuberant granulations usually, but also occasionally by a pterygium-like encroachment of epithelium. (This was noted in the left eye of my first case.) Bullæ may occur in these overgrowths, he states, and the

effects of pemphigus are manifest as far back as the equator of the globe.

v. Marenholtz[55] describes the pathology of pemphigus of the conjunctiva as he found it in sections from the eye of a thirteen-year-old syphilitic. The corneal epithelium was irregularly thickened, and sent epithelial pegs of varying lengths into the corneal stroma. Bowman's membrane was absent entirely, and in the parenchyma of the cornea were numerous round-cells closely packed in rows. In a few fields the corneal stroma had broken down, and the fibers were pushed together and stained badly. Over these fields the epithelium was quite thin and broken in places. (It is probable that this condition obtained in my second case, accounting for the yellow macules in the corneal stroma.)

Adam considers the raised nodules he describes as characteristic of pemphigus, but v. Marenholtz states they were absent in all the sections examined in his case.

Treatment.—Fuchs states that treatment has no power to restrain the process. Arsenic administered internally has usually been the empirical treatment.

In Burk's[11] case with a positive Wassermann, salvarsan was without effect.

Ogiu and Imai's[26] case resisted all therapeutic efforts, even frequent treatments with radium.

In Bordley's[6] case the patient was given salvarsan and sodium cacodylate; the eyes were kept bathed in a solution of zinc sulphate, and from time to time boric ointment was rubbed into the ocular conjunctiva. Later, after all treatment had proved ineffectual, anaërobic cultures, made on human muscles, after the methods of Noguchi and Flexner, resulted in the isolation of a motile bacillus from which a vaccine was made and used. At first high hopes were entertained for the patient's recovery, as he became wonderfully better almost at once, but the disease progressed and death occurred four months after the onset.

v. Marenholtz[35] instilled fibrolysin into the conjunctival sac twice daily for four months, and states that the overgrowths of epithelium were retarded by its use.

Bane[3] states he has had good results in one case with the *x*-ray.

In the case of Le Roux,[22] which is one of the few where bullæ could be demonstrated in the conjunctiva, the cornea was covered with what resembled an onion skin, which was in truth the corneal epithelium detached in large part from the underlying tissues. This yielded with surprising rapidity to treatment with hot compresses, atropin, and yellow oxid of mercury ointment. At the time of writing the cornea showed only slight opacities, which caused but little reduction in the visual acuity, and there had been no cicatricial contraction of the conjunctiva.

Transplantation of conjunctiva, mucous membrane, and even skin has been tried, but all failed, with few exceptions.

In my two cases one, the syphilitic, improved under dionin, atropin, and argyrol locally, with mercury internally, the dionin and mercury probably being responsible for the improvement. My other case steadily became worse in spite of everything that was used.

BIBLIOGRAPHY.

1. Adam, C.: Untersuchungen zur Pathologie des Pemphigus conjunctivæ, *Zeit. f. Augenheilk.*, vol. xxiii.
2. Ballard: Pemphigus, etc., *Med. Times and Gaz.*, 1871, vol. i.
3. Bane, W. C.: Pemphigus., *Oph. Rec.*, vol. xx.
4. Bäumler: Pemphigus conjunctivæ und essentielle Schrumpfung, *Klin. Monatsbl. f. Augenheilk.*, 1885.
5. Bleibtrau: *Berlin. klin. Wochenschr.*, xxx.
6. Bordley, J.: Pemphigus of Conjunctiva, *Oph. Rec.*, vol. xxii.
7. Borysickiewicz: Pemphigus conjunctivæ vulgaris, *Klin. Monatsbl. f. Augenheilk.*, 1879.
8. Bouchart: Pemphigus of Conjunctiva, *Oph. Rev.*, vol. xxx.
9. Bourgeois: Ocular Pemphigus, *Union Med. du Nord-est.*, vol. xxxv.
10. Bowen: Bullous Dermatitis Following Vaccination, *Jour. Cut. and Gen.-Urin. Dis.*, vol. xix.
11. Burk: Pemphigus of Conjunctiva, *Klin. Monatsbl. f. Augenheilk.*, December, 1913.
12. Casali: Ocular Pemphigus, *Annali di Ottalmol.*, vol. xl.

13. Chandler, H. B.: Pemphigus of Conjunctiva, *Amer. Jour. Dermatol.*, April, 1912.
14. Corlett: *Jour. Cut. Dis.*, vol. xxvi.
15. Dahnhardt: *Deutsche med. Wochenschr.*, 1887, No. xxxii.
16. Demme: *Verhandl. d. Cong. f. inn. Med.*, Wiesbaden, 1886.
17. Fehr: Pemphigus of Conjunctiva in Severe Syphilis, *Oph. Rev.*, vol. xxx.
18. Franke: Der Pemphigus and die essentielle Schrumpfung des Bindehautsackes, 1900.
19. Gelpke: Ueber Pemphigus Conjunctivæ, *Klin. Monatsbl. f. Augenheilk.*, 1885.
20. Groenouw: Beziehungen der Allgemeinleiden zu Veränderungen des Sehorgans, Graefe-Saemisch, xi, 1.
21. Howe: Bullous Dermatitis Following Vaccination, *Jour. Cut. Dis.*, vol. xxi.
22. Le Roux: Ocular Pemphigus, *Arch. d'Opht.*, vol. xxxiii.
23. Malcolm, Morris, and Leslie Roberts: Pemphigus der Haut und Mundschleimhaut, verbunden mit essentieller Schrumpfung und Pemphigus der Bindehaut, *Monatsbl. f. prakt. Dermat.*, viii.
24. Matzumota: Pemphigus of Conjunctiva Involving the Mucous Membrane, *Nippon Gank. Zasshi*, October, 1913.
25. v. Michel: Pemphigus der Bindehaut, *Zeitschr. f. Augenheilk.*, iii.
26. Ogui and Imai: Pemphigus Treated with Radium, *Nippon Gank. Zasshi*, November, 1913.
27. Parsons: Pathology of the Eye, vol. i, part i.
28. Pergens: Pemphigus des Auges, 1901.
29. Pernet and Bullock: Acute Pemphigus, *Brit. Jour. Dermatology*, vol. viii.
30. Pokrowsky: Pemphigus of Conjunctiva, *Klin. Monatsbl. f. Augenheilk.*, vol. lii.
31. Sachsalber: Pemphigus Conjunctivæ, *Klin. Monatsbl. f. Augenheilk.*, 1894.
32. Santos-Fernandez, J.: Pemphigus Affecting the Eyes, *Arch. d'Opht.*, vol. xii.
33. Sattler: Präparate von Pemphigus Conjunctivæ, *Bericht über die XII. ophth. Ges. zu Heidelberg*, 1879.
34. Uhthoff: Pemphigus der Bindehaut, *Berlin. klin. Wochenschr.*, 1893, No. 11.
35. von Marenholtz, F.: Ein Beitrag zur Aetiologie, Pathologie und Therapie des Pemphigus Conjunctivæ, *Zeit. f. Augenheilk.*, 1912, vol. xxviii.
36. Weidler, W. B.: Pemphigus of Conjunctiva, Sec. on Oph., Amer. Med. Assoc. Trans., 1912.

DISCUSSION.

DR. W. B. MARPLE, New York: In the New York Eye and Ear Infirmary Reports for 1900 I made a contribution to the literature of this subject, based upon the study of a case of this disease which I had had under observation since September, 1897. The patient was a man of sixty-five who denied syphilis, of which there was no evidence, nor had he ever had general pemphigus. There were some lesions in his throat which our dermatologic consultant pronounced pemphigus.

The eye trouble began in October, 1896, first in the right and then in the left eye, and the condition became gradually

worse. There was entropion of both lower lids, with symblepharon in both eyes below, which slowly progressed. In November, 1898, the left lower lid was completely adherent to the globe at the level of the lower margin of the cornea; there was no culdesac, and the motility of the eye was much restricted. A knobby, opaque mass covered the lower margin of the cornea for about 1½ mm. from the limbus. At this time I separated the lid and globe (which was easily done with scissors) and the next day inserted a Thiersch graft. From a surgical standpoint, the operation for the relief of the symblepharon was a perfect success, but it did not stop the advancing corneal opacity, which, in April, 1899, almost covered the lower half of the cornea. At that time, fortunately, the process in that eye seemed to come to a standstill and made no further appreciable progress while under observation for over a year. Vision was then $\frac{20}{40}$ with + 1.5 D. sph.

In the right eye, where no surgical interference was resorted to, the process progressed, the nasal half of the cornea became covered with dry, opaque tissue, and vision was reduced to hand movements at 10 feet. Several years after this I ran across this patient in the blind ward on Blackwell's Island, and I found that the process in the left eye had subsequently involved the whole of the cornea, so that the prognosis seems to be absolutely hopeless.

On one occasion I showed the case at the Academy of Medicine, and Dr. Herman Knapp stated that, after carefully examining my patient, he understood for the first time the proper interpretation to put upon a condition in a patient which had been puzzling him for some months past; it was the first case of the disease he had ever seen. Such a statement from an observer with as large an experience as Dr. Knapp was certainly convincing testimony as to the rarity of the condition. I have never seen another case since.

DR. WALTER E. LAMBERT, New York: I had the opportunity, some years ago, of seeing a case of pemphigus of the conjunctiva, one of the most desperate cases I had ever seen. The patient, a woman fifty years old, had had a very general eruption over her body, especially on the face, when the lids and conjunctiva became involved. It suggested small-

pox to the physician, and the Health Officer was called in on that account. All the mucous surfaces of the nose and mouth were involved, and the conjunctiva and the cornea were the seat of vesicular pustular eruptions. She had a high temperature and was very sick. I regarded the case as one of pemphigus, and Dr. Elliot made a diagnosis of septic pemphigus. The patient became rapidly worse, and died in four days of general sepsis. The origin of the sepsis was thought to be an infection from a submucous operation in the nose, performed about a week before the beginning of the trouble. Bacteriologic examination showed streptococci.

DR. E. A. SHUMWAY, Philadelphia: I think the point that Dr. Marple has brought out as to the presence of syphilis in these cases is important. I have seen three cases of pemphigus. I saw Dr. Marple's case in the New York Infirmary, and another patient in the Philadelphia Hospital who became blind because of the encroachment of the opacity on the cornea. The third case has been under my care at the German Hospital for three years. In this patient repeated vesicles occurred, which were followed by raw surfaces, and symblepharon gradually resulted. I had the throat and nose examined, and lesions were found there; but the rhinologists thought that they were of a syphilitic character, and she afterward developed septal perforation. We had a Wassermann test made, and it was found positive. She was put on antisyphilitic treatment, and it is my impression that she improved while she was taking the treatment. Fewer plaques were seen, and fewer raw places. The progress of the case has been slow, but she now has almost complete adhesion of the lower lid to the bulbar conjunctiva. The cornea is still fairly clear.

The question, then, of syphilis in these conditions is important to bear in mind. There seem to be different types of the disease, and when syphilis occurs and there is a positive reaction, it is important to give mercurial treatment.

Fig. 1.—Before operation. Showing the extreme ptosis of the left upper eyelid and small palpebral fissure. The right eye inturning, with the dense leukoma.

Fig. 2.—Eversion of the upper lid, showing the size of the growth.

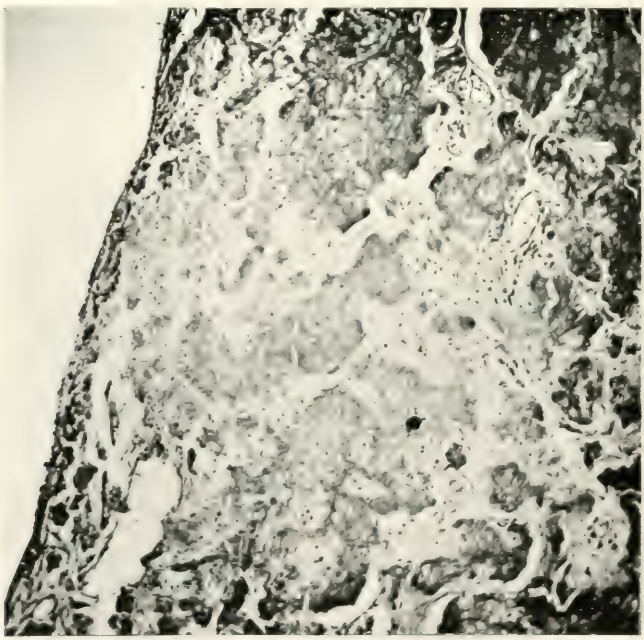


Fig. 3.—Photomicrograph showing atrophy of the conjunctiva and the underlying masses of the amyloid material. (Zeiss Obj. A A, Oc. 2.)

REPORT OF A CASE OF COLLOID DEGENERATION OF THE UPPER LID.

ARTHUR J. BEDELL, M.D.,
Albany, N. Y.

Colloid, hyaline, or amyloid degeneration of the conjunctiva or eyelid is a very rare condition. In 1878 Bull read a paper before this Society citing a case of eyelid and orbit involvement. Prout and Bull, in 1879, recorded another experience. No others are found in American literature, and only one in English—that by Herbert. Oettingen, in 1871, first described the lesion in a communication published in Dorpat. His report was followed by many other observations, most of which were in Russia and Galicia. The appended bibliography, compiled after an exhaustive search, seems to cover all previous experiences.

Mr. J. J. D., aged forty-eight years, a strong, vigorous lumber inspector, was first seen February 27, 1915. While working on cypress, about two years ago, he first felt something in his left eye. He always thought that some sawdust had gotten in the eye, because a doctor was said to have removed a particle from it. His wife, however, thought that the left upper lid had been drooping for at least two years, so that probably he had conjunctival irritation even then. Wassermann negative. Blood negative.

Right eye, V. = fingers two feet. Conjunctiva of both lids and globe absolutely negative. Palpebral fissure 10 mm., the eye turned decidedly inward 30 degrees, the result of paresis of the external rectus. A dense corneal opacity, about 5 mm. in diameter, with very faint superficial vascularization, was slightly to the nasal side and below the apex. Pupil 3.5 mm., active to light and accommodation; anterior

chamber of normal depth. A 1 mm. anterior polar opacity of the lens was the only other abnormality.

Left eye, V. = $\frac{20}{30}$, type 10. Complete ptosis, with a 3 mm. palpebral fissure on extreme elevation (Fig. 1). The upper lid was rounded out by a firm mass of cartilaginous consistence, which was not attached to the skin, but formed an integral part of the conjunctiva and tarsus.

On eversion of the lid, the growth, 35 x 25 x 12 mm. in size, rolled out (Fig. 2). The entire tumor was covered by smooth, partly adherent conjunctiva, except an oval area 25 x 8 mm., which formed its anterior portion. Over this the conjunctiva was very rough, with large, dark-red granulations, which projected over the free tarsal conjunctiva, suggesting one form of tuberculosis.

The uninvolved conjunctiva, which included one-half of the upper tarsal, the entire bulbar, and lower palpebral conjunctiva, was negative in detail, showing no congestion, no follicular enlargement, and no signs of old or new trachoma. Pupil 4 mm., active to light and accommodation; media clear; disc distinctly outlined, with a small central excavation; no vessel change.

On March 1, 1915, under ether, the mass on the upper lid was dissected out, cutting through the posterior one-half of the tarsus, and working beneath the skin and conjunctiva. A complete enucleation of the growth was possible. The conjunctival surfaces were approximated with three silk sutures, and the eye firmly bandaged. Very little reaction followed operation, and the sutures were removed on the sixth day. Eyelid motion has been steadily regained, until now there is good action, and the conjunctiva wound is smooth. V. sph. + 1.25 D. = $\frac{20}{15}$; with a + 1.75 added type 1.

PATHOLOGIC REPORT BY ELLIS KELLERT, M.D., DIRECTOR OF THE BENDER HYGIENIC LABORATORY.

The specimen consists of a flattened, reddish-gray mass of tissue, almost circular in outline, measuring 3 x 2.5 x 0.6 cm. One half of the section is thicker and firmer than the remaining portion, which is thin, flat, and soft in consistence. The

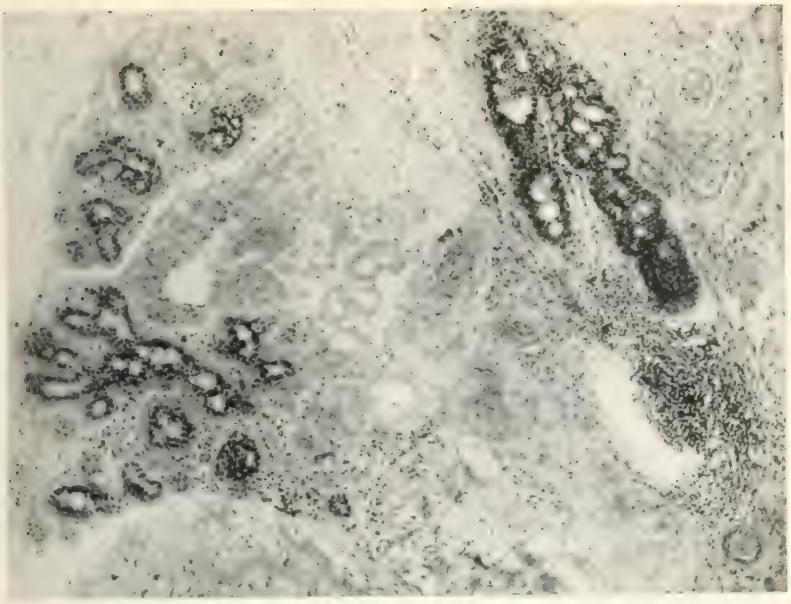


Fig. 4.—Photomicrograph—Meibomian gland partially degenerated. (Zeiss Obj., A A, Oc. 2.)

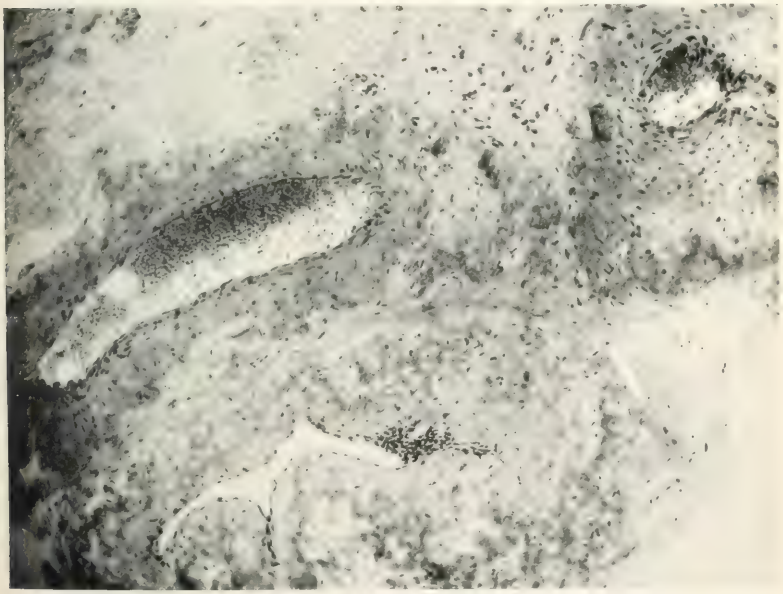


Fig. 5.—Photomicrograph showing vascular changes. (Zeiss Obj. A A, Oc. 2.)

surfaces are somewhat uneven, pitted, and covered by a slight mucoid exudate. On sectioning the specimen gritty material is encountered in the thickest portion of the mass. The cut surface is grayish-brown, mottled, and translucent. A rare but prominent blood-vessel is seen. The margin of the thickest portion presents a glistening, grayish-streaked appearance.

Microscopic sections stained with hematoxylin and eosin show the specimen to consist almost entirely of pink-staining, structureless masses, varying greatly in size. Beneath the epithelium the masses are smallest, becoming larger and conglomerate in the center of the tumor. There are also found within the pink-staining bodies beneath the epithelium many irregular, dense blue areas, suggesting deposits of calcareous material. Small and isolated strands of connective tissue can be seen, particularly about the blood-vessels. Cellular elements are scattered throughout the section, and there is an occasional focal accumulation of lymphocytes containing a giant-cell. The condition appears to be that of amyloid change or degeneration.

The conjunctival epithelium is somewhat atrophic, and at certain points entirely absent, exposing the underlying amyloid masses (Fig. 3). These are quite sharply outlined, but present no regular pattern. They stain very unevenly, probably because of the varying concentration of the amyloid material. Many of the bodies have a pale periphery, while others have lighter centers. The largest bodies stain most deeply with the eosin; some are seen to consist of numerous small fragments, but in others the fusion is complete, forming a large mass of amyloid material. The periphery of certain paler and apparently more recent accumulations exhibits rounded or flattened nuclear bodies, which may be surrounded by a trace of cytoplasm. They appear to be adherent connective-tissue cells or cells thickened with an accumulation of the amyloid material. Within many of the masses, particularly the larger ones, are small, rounded, bluish bodies which vary in size from an object visible only with the highest power of the microscope to globules as large as lymphocytes. They may be found isolated or in clusters without regular distribution; some are nucleated and resemble cellular structures. The smaller ones are perfect

circles, but the larger bodies are irregular in outline. These objects appear to be the earliest stages of lime deposition. Interspersed among the amyloid masses are remnants of altered connective tissue. These bands are found in greatest abundance about the sebaceous glands. Elsewhere they may be seen encircling or blending imperceptibly with the amyloid substance. Nuclei are abundant in these areas, but few cellular outlines are seen. Scattered throughout the sections are many lymphocytes and plasma cells, a few distorted polymorphonuclear leukocytes, and an occasional mast-cell. Many of the larger masses lie in irregular spaces, having smooth surfaces, but no endothelium, although an occasional endothelial-like cell is found. Some of the spaces present appear to be formed by the contraction of the amyloid substance.

The blood-vessels are markedly altered. The walls are thickened and composed largely or entirely of amyloid bodies. The intima is little changed, or shows an increased number of nuclei. The endothelium in general is somewhat swollen and hyperplastic, and occasionally forms a syncytial mass within the lumen. The individual coats cannot be recognized, nor can the muscle layers be distinguished, so complete is the degeneration. They are recognized as blood-vessels only by their shape and the presence of erythrocytes within the lumen. The walls of the vessels contain many nuclei irregularly distributed. Those vessels that are not completely degenerated have deposits of amyloid in the adventitia and outer layers of the media, suggesting that these are the earliest portions of the vessel-wall to be attacked. An occasional vessel is fissured, and blood-cells can be seen escaping into the surrounding tissues. A few thin-walled structures which appear to be lymphatics contain within their lumina masses of the amyloid substance.

The sebaceous glands appear normal. The connective tissue surrounding the glands is increased in amount, and only moderate diffuse degeneration is present, but none of the isolated amyloid masses found elsewhere in the specimen occur. The Meibomian glands show advanced changes. The acini are encroached upon, and in many instances replaced by the amyloid material. The lumina are contracted or filled with pink-staining colloid containing few cellular

elements. Many of the acini are distorted and devoid of epithelium, the remaining structure consisting of homogeneous material. Certain lobules are completely degenerated, and only shadowy outlines of glands can be seen, with an occasional poorly stained epithelial cell. The stroma is similar in appearance and staining to the amyloid substance elsewhere in the section.

A number of staining methods were tried. Dilute aqueous methyl-violet colors the amyloid material violet to blue, the violet being most pronounced in the region of the blood-vessels. Other tissues stain blue. After treatment with Lugol's solution the masses appear light brown, the color being greatly deepened upon the addition of 5 per cent. sulphuric acid. The method of Birch-Hirschfeld gave the most clear-cut results. The connective tissue stains brownish, while the amyloid masses appear dull red. Other special stains gave indifferent results, and in no case was the staining reaction permanent. The most persistent were the methyl-violet and the Bismarck-brown-methyl-violet method of Birch-Hirschfeld, but all tended to fade after a few days. Sections stained by the Gram-Weigert method and for tubercle bacilli were all negative.

REFERENCES.

Becker: Amyloid Degeneration of Tarsi, *Finska Lakaresallskapets Handlingas*, vol. xvii, pp. 150–158.

Bull: A Case of Amyloid Infiltration of the Eyelid and Orbit, with Remarks, *Medical Record*, 1878, p. 266.

Bushnelli: Caso di degenerazione amiloido del tessuto-peritarsale, *Annali di Ottalmologia*, No. x, p. 532.

Colucci: Di un tumore ialino-amiloideo della congiuntiva, *Annali di Ottalmologia*, 1900, p. 349.

Dimmer: Ein Fall von hyaline Degeneration der Lider und der Conjunctiva, mit ausgebreiteter Verkalkung und Verknocherung, *Zeit. für Augenheilkunde*, 1903, p. 474.

Del Monte: Contributo allo studio della degenerazione amiloide della congiuntiva e delle palpebre con speciale riguardo a taluni reperti non ancora conosciuti, *Archiv. di Ottalmologia*, August, 1910, p. 57.

De Wecker and Masselon: Degenerescence amyloide de la conjonctive, *Manuel d'Ophtalmologie*, Paris, 1889, p. 148.

Ewetzky: Ueber hyaline Degeneration der Augenbindehaut, *Moscow Ophthal. Soc.*, 1893, Westnik Oph., xi, p. 192.

Guth: Ein Fall von hyaliner Entartung der Bindehaut, *Centralblatt für prakt. Augenheilk.*, 1902, p. 386.

Herbert: Colloid Degeneration, *Oph. Soc. United Kingdom*, 1902.

Hippel: Ueber amyloide Degeneration der Lider, *Graefe's Arch. für Aug.*, 1879, vol. xxv, p. 1.

Hubner: Zur amyloiden Erkrankung der Bindehaut, *Beitrag zur Augenheilk.*, 1899, p. 1.

Ishihara: Ein Beitrag zur Entwicklung der amyloid Degeneration der Konjunctiva, *Klinische Monatsblätter für Augenheilkunde*, 1913, p. 65.

Ischreyt: Ueber hyalin Degeneration der Conjunctiva, *Arch. für Augen.*, 1906, p. 400.

Jakowlewa: Hyaline Thromben in den Gefassen der Aderhaut, St. Petersburg. Oph. Gesel., January, 1914, p. 729; *Klinische Monatsblätter für Augenheilkunde*, 1914.

Kamocki: Ein Beitrag zur Kenntniss der hyalinen Bindehaut-Entzündung, *Centralbl. für prakt. Augen.*, 1886, p. 68.

Kamocki: Untersuchungen über hyaline Bindehautentartung, *Beiträge zur Augenheilk.*, 1893, p. 1.

Kolominsky: Ein Fall von hyalin-amyloider Degeneration der Konjunctiva, *Klinische Monatsblätter für Augenheilkunde*, 1912, p. 559.

Kruch and Fumagalli: Degenerescence amyloide de la conjonctive, *Annales d'Ocul.*, 1895, p. 39.

Krudener: Ein Beitrag zur pathologischen Anatomie der Amyloidtumoren, Dorpat, 1892.

Kyber: Studien über die amyloide Degeneration, Dorpat, 1871.

Kyber: Weitere Untersuchungen über die amyloide Degeneration, *Virchow's Arch.*, No. 8, 880, p. 1.

Kyll: Die amyloide Degeneration der Conjunctiva, Bonn, 1876.

Leber: Ueber amyloide Degeneration der Bindehaut des Auges, *Graefe's Arch. für Oph.*, 1873, p. 163.

Leber: Ueber die Entstehung der Amyloidentartung vorzugsweise nach Untersuchungen an der Bindehaut des Auges und über die Herkunft der Amyloidkörperchen in der atrophischen Nervensubstanz, *Graefe's Arch. für Oph.*, 1879, p. 257.

Leber: Die Conjunctivitis petrificans, *Graefe's Arch. für Oph.*, 1900, p. 1.

Legrain: Hypertrophie et degenerescence amyloides des cartilages tarses probablemenent d'origine heredo-syphilitique, *Recueil d'Ophtalmologie*, 1904, p. 695.

Mandelstamm and Rogowitch: Ein Fall von Amyloid der Conjunctiva bulbi et palpebrarum, *Graefe's Arch. für Oph.*, 1879, No. 25, p. 248.

Morax and Landieu: Degenerescence hyaline du tissu sous conjunctival et epitarsien des deux paupieressuperiuires, *Annales d'Oculistique*, 1912, p. 25.

Michel: Das tarsale Bindegewebe kann von einer fettingen und von einer amyloiden Degeneration befallen werden, *Graefe-Saemisch Handbuch der Augenheilkunde*, vol. v, p. 336.

Monesi: Sopra un caso di degenerazione ialina della congiuntiva, *Archiv. di Ottalmologia*, 1900–1901, vol. viii, p. 289.

Mooren: Ophthalmologische Mittheilungen, Berlin, 1874, p. 27.

Oettingen: Die ophthalmologische Klinik Dorpats in den drei ersten Jahren ihres bestehens, *Dorpater medizinische Zeitschrift*, 1871, p. 1.

Poppen: Degenerescence amyloide de la conjonctive et du cartilage tarsien, *Annales d'Oculistique*, 1913, p. 108.

Poriwoew: Ein Fall von hyalin-amyloider Geschwulst der Conjunctiva, *Jahresbericht über Ophthalmologie*, 1886, Westnik Oph., iii, 9, p. 182.

Poscharisky: Ueber amyloide Geschwulste der Conjunctiva bulbi, *Med. Obosr.*, lx, M. 19.

Puccioni: La malattia amiloide delle palpebre e della congiuntiva, *Annali di Ottal.*, 1898, p. 215.

Prout and Bull: Sarcoma of the Tarsus and Conjunctiva of the Lid with Amyloid Degeneration, *Arch. Oph.*, 1879, p. 73; *Arch. für Augenheilkunde*, 1879, p. 221.

Quaglino-Guaita: Contribuzione alla storia clinica ed anatomica dei tumori intraed extra oculari, Tumore della palpebra inferiore destra da degenerazione amiloide della congiuntiva edel tarso, *Annali di Ottal.*, 1877, p. 163.
Raehlmann: Ueber amyloide Degeneration der Augenlides, *Arch. für Augenheilkunde*, 1881–83, p. 402.
Raehlmann: Ueber hyaline und amyloide Degeneration der Conjunctiva des Auges, *Virchow's Arch.*, 1882, No. 87, p. 325.
Raehlmann: Ueber amyloide Degeneration der Lider und Konjunktiva des Auges, *Klinische Monatsblätter für Augenheilkunde*, 1905, p. 435.
Reymond: Contribuzione allo studio delle congiuntiviti, Degenerazione amiloidea della congiuntiva, *Annali di Ottalmologia*, iv, p. 349.
Rumschewitsch: Ueber die hyaline und amyloide Entartung der Bindehaut, *Arch. für Augenheilkunde*, 1892, p. 363.
Rogman: Un cas de degenerescence hyaline des paupieres, *Annali d'Oculistique*, 1898, p. 89.
Sabrazes and Gasau: Colloide du tissu conjonctif et des cellules geantes dans un cas d'enorme hyperplasie conjunctivale des paupieres suite de trachome, Reunion Biologique de Bordeaux, February 7, 1912, *Compt. Rendus Hebdomadaries*, 1912, p. 241.
Schieck: Ueber die hyalin und Amyloiderkrankung der Conjunctiva, *Graefe's Arch. für Oph.*, 1907–08, p. 119.
Schmidt: Ueber die Beteiligung des Auges an der allgemeinen amyloid Degeneration, *Centralbl. für all. Path.*, 1905, p. 49.
Scimemi: Delle Relazioni fra la sostanza iolina e e'amiloidea nei tumori iolinoamiloidei della congiuntiva, XI. International Congress, Rome, 1894.
Steiner: Ein Fall von amyloider Degeneration der Bindehaut und des Tarsus bei einem Malayen, *Centralbl. für Augenheilkunde*, February, 1904, p. 3.
Stroehmberg: Ein Beitrag zur Casuistik der amyloiden Degeneration an dem Augenlidern, Dorpat, 1877.
Tschemolosow: Zur Frage der hyalinen Degeneration der Bindehaut, *Jahresbericht über Ophthalmologie*, p. 189.
Uhthoff: Ein Fall von ungewöhnlicher Degeneration der menschlichen Conjunctiva, *Virchow's Archiv.*, 1881, No. 86, p. 322.
Van Duyse: Deux cas de degenerescence hyaline de la conjunctiva, *Arch. d'Oph.*, 1894, p. 742.
Venneman: Les boules hyalines ou "colorables" dans les tissus de la conjonctivi enflammee, Société Française Ophtal., vol. x, p. 364.
Vogel: Ueber Perichondritis des Tarsalknorpels, Bonn, July 19, 1873.
Vossius: Ueber hyaline Degeneration der Conjunctiva, *Beiträge zur path. Anat. und zur allgemeinen Pathologie*, 1889, p. 293.
Wichman: Die Amyloiderkrankung, *Beitrag zur path. Anat. und zur allgemeinen Path.*, 1893, p. 487.
Wild: Beitrag zur Kenntnis der amyloiden und der hyalinen Degeneration des Bindegewebes, *Beiträge zur pathologischen Anatomie und Physiologie*, 1884–86, p. 177.
Zwingmann: Die Amyloidtumoren der Conjunctiva, Dorpat, 1879.

A CASE OF EPIBULBAR SARCOMA.

HOWARD F. HANSELL, M.D.,
Philadelphia, Pa.

Mrs. M. K., aged forty-six years, was admitted to the Jefferson Hospital November 10, 1914; service of Dr. J. H. Gibbon. She complained of pain in both legs, and after examination the provisional diagnosis of infectious thrombophlebitis was made. No family history of renal, cardiac, tubercular, or malignant disease was obtainable. The patient has had three miscarriages, the last twelve years ago. She had dulness over the right apex and base, and examination of sputum showed a few streptococci, staphylococci, and diplococci, but no tubercle bacilli. Physical examination revealed no conditions relevant to the ocular tumor.

I was asked to see the patient December 31, 1914, because of the presence of a tumor on the left eye. The tumor was round, flesh colored, covered by loosely connected conjunctiva, about the size of a marble, resistant to pressure, but not particularly hard, and situated on the outer, upper quadrant of the sclera, its anterior border about 8 mm. from the limbus. Vision was not affected, and the only subjective symptom was that of annoyance from the presence of the tumor. The media were clear and the fundus healthy. Under local anesthesia it was removed entire. Its base was not only firmly attached to the sclera, but was a part of it. The conjunctiva was sutured and light pressure applied. Healing followed without complication. On May 29th, the day of her last visit, the site of the tumor was marked by a round, smooth area, 3 mm. in diameter, rather dark, but apparently not actually pigmented. There was no sign of recurrence. V. = $\frac{20}{20}$. The eye appeared to be perfectly healthy.

Pathologic Report by Dr. E. D. Funk.—Specimen is a small, semisolid, reddish-yellow mass, measuring 0.5 by 0.2 cm.;

A case of epibulbar sarcoma.

weight, 0.5 gm. The mass cuts easily, and the incised surfaces are grayish-yellow and smooth. The mass was fixed in absolute alcohol, embedded in paraffin, sectioned, and sections obtained with hematoxylin, eosin, and van Gieson's mixture.

Histology.—The sections are made up principally of a dense cellular collection supported by a scanty amount of fibrous stroma. Among the small, round, deeply staining cells appear a number of young blood-vessels, and in areas extensive hemorrhage is present.

Diagnosis.—Small round-cell sarcoma.

The title of epibulbar tumor has been applied to all kinds of growths of the conjunctiva, corneal limbus, and cornea. It does not signify the character, but only the position, of the tumor. The majority have proved to be sarcomata, and, like sarcomata in other parts of the body, are prone to recurrence. Verhoeff and Loring* report 73 cases, comprising the published cases up to that year.

L. W. Crigler† added a new case and abstracted 12 others from the more recent literature. These authors agree in their conclusions, which may be summarized thus: The majority of cases occur in persons of advanced years; they are, as a rule, melanotic; their tendency is not to penetrate the globe, but to recur locally when removed and to produce metastasis. Koller's‡ two cases are interesting, as bearing upon the liability to recurrence. In a man of thirty-eight excision was followed by recurrence, local metastasis, and contact tumors, yet after the removal of the last of these there has been no recurrence for nearly eight years. The second case, a girl of seventeen, had a very small tumor which was removed; recurrence in two and one-half months. This was destroyed with the cautery and no recurrence in six years.

When the sarcoma is limited to the outer membranes of the eye, early and thorough excision, with cauterization of the

* *Arch. of Ophthal.*, 1903, xxxii.

† *Ibid.*, January, 1915.

‡ *Ibid.*, July, 1912.

base (Koller), may be curative, but involvement of the intraocular tissues, recurrence, or rapid growth is an indication for enucleation.

MULTIPLE MELANOTIC NEVI OF CONJUNCTIVA, WITH MALIGNANT INVASION OF THE CORNEAL LIMBUS.

F. P. CAPRON, M.D.,
Providence, R. I.

The following case was first brought to my notice October 17, 1911, at which time Mrs. W., aged forty-one, consulted me for glasses. At that time the condition of the left eye attracted attention on account of slight ptosis and some peculiar brownish pigment deposits on the palpebral and ocular conjunctiva, but as no trouble of any sort had been experienced with that eye, and the spots had existed for eight years with no change, no weight was attached to their presence.

The patient had been a victim of infantile paralysis, and the left side was not developed to an equal degree with the right; also, the left foot dragged somewhat in walking, making an appreciable limp. V. O. D., $\frac{20}{40}+$, sph. $-$ 1.50 D. $= \frac{20}{20}$; O. S., $\frac{20}{50}$, sph. $-$ 1.25 D. $= \frac{20}{20}-$. Gave $-$ 1.25 O. D., $-$ 1.00 O. S., for distance. Reading ability satisfactory without glasses. Fundi and muscles normal.

Soon after this visit Mrs. W. and family went abroad for a year or two, and was not seen again until December 4, 1914, more than three years later, when she gave the following history: While in Jena, about one year after consulting me, she discovered a tumor on the left globe, extending well down toward the bottom of the culdesac, about $\frac{3}{4}$ inch long by $\frac{1}{3}$ inch in diameter, shaped like a kidney-bean. Dr. Stock, of Jena, was consulted, who at once pronounced the growth malignant, and advised immediate removal. This unexpected state of affairs naturally caused considerable anxiety and the desire to consult other authority. Consequently Dr. Rindfleisch, of Würzburg, was sought out, and, as his opinion was the same, although expressed in a more feeling

manner, the patient yielded to demand for operation, which was done the following day. Healing was uneventful, and the patient experienced no further difficulty. Dr. Rindfleisch gave a pathologic report, calling the tumor a strongly pigmented melanosarcoma rich in cells. Describing the tumor, he stated that the growth rested freely movable upon the conjunctiva, and was in no way connected with the sclera. A sufficient rim of sound conjunctiva was removed with the growth. He considered the prognosis not unfavorable, since the pigmented condition had existed nine years unchanged; also because the growth was not in direct relation with the uveal tract, but warned that any irritation of the pigment spots might develop increase of cell structure and growth of a tumor; also, since the bulbar surface showed several pigmented spots, it was possible that one or more tumors might develop in consequence. Therefore he advised repeated examinations by an oculist. He gave the vision at that time O. D., sph. $-$ 0.75 D. $= \frac{6}{6}$; O. S., sph. $-$ 1.25 D. $= \frac{6}{9}$. The operation was normal in every way, and there were no enlarged glands about head or neck.

At the time of this second visit to me in December, 1914, Mrs. W. complained of irritation of the left eye, which was somewhat congested and showed superficial ulceration of cornea at upper sclerocorneal margin. There was some apparent increase in pigmented spots. V. O. D., $\frac{20}{40}$; sph. $-$ 1.50 D. $\bigcirc$ cyl. $+$ 0.25 D. axis 90° $= \frac{20}{20} -$; O. S., sph. $-$ 1.75 D. $= \frac{20}{30} +$. Ordered atropin and cocain, also dionin.

The ulceration gradually healed, but was followed by a similar condition at the outer sclerocorneal margin, which in time gradually healed, only to be succeeded by ulceration at the inner margin, then the inferior sclerocorneal border. All healed after a while with the exception of the inferior ulceration, which became somewhat deep and painful. During the attempt at healing of this portion an elevation of the surface gradually appeared, eventually developing into a dark brownish tumor. In spite of all treatment considerable congestion, accompanied by decided pain and discomfort, prevailed. In all places where ulceration had formed pigment deposits appeared.

February 10, 1915, V. O. D. $= \frac{20}{24} -$; O. S. $= \frac{20}{70} -$. Own glass V. O. D. $= \frac{20}{160} -$. No improvement of O. S. by

any glass. From the rather rapid development of this growth at the inferior margin, it became evident that there was little or no hope of disposing of the trouble by treatment, and enucleation appeared to be demanded.

As it seemed wise to seek advice, Dr. Derby, of Boston, was asked to see the patient; later Dr. Greenwood also saw her, together with Dr. de Schweinitz, all of whom agreed with the writer in the matter of operation. Some time elapsed before the patient was again seen, so that when she presented herself on March 2d to arrange for enucleation the following day, a most decided change for the worse was manifest in the condition of the globe. There was much more congestion, increase of pain, and great reduction in vision, all accompanied by a disturbing loss of self-control.

On March 3d the eye was removed, together with the larger portion of the pigmented conjunctiva, two spots being left to be cared for later as occasion demanded. There seemed to be some danger of extreme cicatrization, which would interfere with the use of an artificial eye, and much depended upon this in the mind of the patient, while to the operator it seemed reasonable to believe the portion left could be more satisfactorily removed after healing, either by operation or treatment.

The operation was attended by no untoward circumstance save profuse hemorrhage into the cellular tissue, rendering the lids perfectly tense and enormously enlarged, undoubtedly due to abscising the nerve far back behind the globe, which was done for the purpose of having a generous amount for pathologic examination if required. Healing was normal, and an artificial eye was inserted in about two weeks.

After about a month it seemed wise to try radium treatment for the patches, and this has been followed faithfully by Dr. Williams, of Boston, up to the present time, with most satisfactory results, and the patient is now well restored to her former physical condition, and the two spots have now

so faded out that they appear much like shadows of their former selves. The one on the edge of the lower lid, near the canaliculus, is only dimly seen, while the other, near center of the stump, a little toward the temporal side, appears much like a dark line.

Dr. Verhoeff made a pathologic examination of the globe, and reported that the tumor did not anywhere invade the interior of the eye, which was perfectly normal. On the cornea the tumor had penetrated only to a point between Bowman's membrane and the epithelium, excepting near the limbus, where it had broken through Bowman's membrane and had begun to invade the corneal stroma.

It seems reasonable to suppose that the corneal implications were due to implantation of cells from the melanotic spots upon the conjunctiva, such a condition as Dr. Rindfleisch may have had in mind, as suggested by his report.

The removal of the globe and the dissipation of the patches left behind, together with the greatly improved condition of the entire stump, should well remove all possibility of further trouble in the light of the fact that there was no extension of the growth much beneath the ocular surface, and also because of entire absence of any symptoms pointing to a constitutional implication.

This unusual formation of melanotic deposits, their long quiescent state, then the rapid development of a moderately large tumor, its complete and easy removal with no disturbance following until three or more years subsequent, all seem to warrant a report of the case.

DISCUSSION.

DR. G. E. DE SCHWEINITZ, Philadelphia: Owing to the courtesy of Dr. Capron, Dr. Derby, and Dr. Greenwood, I had the pleasure of examining this patient. In order to decide whether it was worth while to try radium before proceeding to a radical operation, I submitted the history, which Dr. Capron has read, to two of the best radium ex-

perts in the country, and asked them whether they would consider it justifiable to use radium, or whether they believed it was better to perform an enucleation. Both the experts declined to accept the responsibility of radium treatment. They believed the procedure which was afterward followed to be the only safe one.

REPORT OF TWO CASES OF METASTATIC CARCINOMA OF THE CHOROID AND ONE CASE OF MYXOSARCOMA OF THE ORBIT.

JOHN E. WEEKS, M.D.,
New York City.

CASE 1.—Mrs. S. H., aged sixty-four years, came under observation January 29, 1915. Patient fairly well nourished. Never any severe illness. Two and a half years ago was operated on for carcinoma of the right breast. Healing was satisfactory, but within a few months there were some evidences of recurrence in the form of a few nodules along the cicatrix (reported by her son-in-law, who is a physician).

About December 25, 1914, patient saw flashes of light to the right side, evidently due to a disturbance of the retina of the right eye. The sight of this eye gradually failed. There was very little pain. The vision of the left eye had never been good.

The patient consulted Dr. C. H. May, who diagnosed intraocular neoplasm, probably a metastatic carcinoma, and advised enucleation.

When seen by me the vision of the right eye with correction of the error of refraction was $\frac{2}{4}\frac{0}{0}$; left eye, $\frac{2}{3}\frac{0}{0}$ —. The interior of the right eye showed detachment of the retina to the nasal side of the disc, which was quite sharply defined and rounded to the temporal side and below. The retina at a short distance from the disc where the elevation was most marked was raised $2\frac{2}{3}$ mm. above the normal level. The detachment passed gradually into normally placed retina at the periphery. Tension normal. Slight injection of ocular

conjunctiva, more marked at the nasal side. The left eye showed no abnormity. There was a slightly mottled area near the temporal margin of the detachment, a little deeper in color than the surrounding retina, but no vascular system not belonging to the retina could be detected. Ordinary transillumination was negative.

A diagnosis of intraocular neoplasm, probably metastatic carcinoma, was made, but enucleation was not advised at the time, for the following reasons:

First, it was thought possible that the mass might not be malignant.

Second, if the mass was carcinoma, its growth might be so slow that the patient might pass away before the growth produced painful symptoms, since statistics have shown that the average length of life after the occurrence of metastatic carcinoma in the eye is six to seven months (Oatman).

It was, of course, recognized that the neoplasm might be sarcoma, in which case immediate enucleation would be indicated.

After the first visit I wrote the following letter to the patient's son-in-law:

The patient has a detachment of the retina of the right eye, presenting an oval, slightly raised patch at the nasal side of the optic disc. There is an exudation beneath the retina at this point which renders it impossible to get more than a surmise of the underlying mass. Tension of both eyes approximately normal. I am of the opinion that there is a new-growth beneath the retina at the point mentioned, and from the slight elevation, coupled with the history of carcinoma in another part of the system, I think it very probable that it is a metastatic carcinoma of the choroid. There is, of course, a possibility that it may be a flat sarcoma. When metastatic carcinoma appears in the choroid, the individual is short lived (average, six to seven months in 30 cases collected by Oatman), as this is an evidence of a tendency to general metastasis. In metastatic carcinoma there is little tendency to rapid involvement of the sclera, and extension

along the optic nerve to involve the fellow-eye has not been observed.

In view of the facts that the patient is not suffering pain, the optic nerve is not encroached upon; that if it is a metastatic carcinoma, enucleation would not prolong life, and that there is little or no danger to the fellow-eye from the affected eye, I would advise against enucleation at present.

The mass should be watched, and if it grows rapidly or causes pain, or if it becomes probable that it is sarcoma at any time, the eye should be removed.

On February 15th the vision of the right eye was $\frac{20}{50}$, the detachment of the retina had increased, and the injection of the ocular conjunctiva was more marked. There was also a very slight increase in tension and some neuralgic pain. As it was evident that the growth was increasing rapidly, I advised enucleation, which was performed February 16th. The hemorrhage and edema following enucleation were much greater than usual; healing proceeded slowly, but favorably; the resulting socket was smooth and free from complications. An artificial eye was inserted March 23, 1915.

The microscopic examination discloses carcinoma, which, of course, is metastatic.

June 28th, patient's condition excellent. Very slight advance in growth of nodules at side of wound in breast.

CASE 2.—Margaret S., aged forty-six years, came under observation May 23, 1902, complaining of loss of vision in the right eye. Examination disclosed anterior chamber shallow. Vision greatly reduced. Detachment of retina outward and below sharply defined, except at the temporal border. Elevation, 5 mm. A brownish mass could be detected beneath the retina, but there was no independent system of blood-vessels discernible. No pain. Some injection of the ocular conjunctiva in the outer lower quadrant. There was no history of previous illness or of a neoplasm in other parts of the body.

A diagnosis of intraocular neoplasm was made, and enucleation advised. Operation May 25, 1902, followed by un-

Case of myxosarcoma of orbit.

eventful healing. On microscopic examination the growth was found to be carcinoma. The arrangement of the cells and alveoli suggested adenocarcinoma.

How such a growth could appear without appreciable carcinoma in some other part of the system was a mystery. Three months later the patient died at Bellevue Hospital. Autopsy revealed carcinoma of the right lung. This finding, of course, explained the occurrence of the metastatic growth in the choroid. I do not know of a similar case reported.

Metastatic carcinoma of the choroid is almost invariably a late complication of carcinoma. Affection of both eyes occurs in approximately one-third of the cases.

In regard to treatment, I am of the opinion that enucleation should be resorted to only when distressing symptoms are the result of the growth, as enucleation cannot lengthen life or prevent occurrence in other parts of the system.

CASE 3.—Howard H., aged six and a half years, was referred to my clinic at the New York Eye and Ear Infirmary by Dr. Frank S. Skeel. Patient fairly well nourished; pale. Six weeks ago the outer margins of the upper lid of the right eye became swollen, red, but not tender. The swelling gradually increased, causing drooping of the lid and pushing the eyeball downward and slightly forward. The swelling was elastic, and gave the impression of a tense abscess on palpation. Three weeks before coming to the Infirmary the swelling was incised by the family physician, but no pus was discovered. At the time of admission the condition described above was much accentuated. No pain of moment. On January 28th the eruption of measles developed, and the child was sent to the Willard Parker Hospital. Case readmitted to the Infirmary February 4, 1915. The swelling had markedly increased and presented the appearance as shown in the accompanying photograph. There was no enlargement of the preauricular, submaxillary, or cervical glands.

On February 5, 1915, exenteration of the orbit. The operation consisted in the removal of the entire mass to the apex of the orbit, together with the periosteum and as much of the

tissue of the eyelids as was involved. A small amount of tissue was left at the very apex of the orbit, because of the difficulty in excising it and fear of excessive hemorrhage. The skin of the lid was turned in against the orbital wall and held in position by gauze packing. Healing progressed satisfactorily and granulation tissue developed under rubber tissue dressing until March 2d, when the granulating surface was covered with Thiersch grafts. The surface, with the exception of a small area at the nasal side, was soon dermatized.

The patient gained in weight and improved generally until about June 12th, when he became mentally depressed and began to lose weight. At the present time there is a small nodule at the apex of the orbit which is soft and of a dark, bluish-red color, evidently indicating a return of the growth. Microscopic examination shows the growth to be myxosarcoma.

Parsons writes of myxosarcoma as edematous, small-cell sarcoma. Occurrence is most frequent in early life, although tumors of this variety have been observed in advanced adult life. Recurrence after removal has been reported in approximately two-thirds of the cases, and a fatal issue in about the same percentage.

DISCUSSION.

DR. C. H. MAY, New York: I wish to say a word about the first case, the patient that Dr. Weeks alludes to as seen by me. As he states, it was impossible to say whether the growth was a metastatic carcinoma in a person who had had carcinoma of the breast, or a sarcoma, which it might have been. I considered it wiser to enucleate the eyeball. I sent the patient in consultation to Dr. Callan, who agreed with me that, although there might be no danger in waiting, it was wiser to remove the eyeball. I am glad to hear that the woman apparently suffered in no way from the delay in enucleation.

DR. S. C. MAXSON, Utica, N. Y.: I think that it is important, in cases of sarcoma of the choroid, to perform a radical operation early. I saw a case of a woman, about sixty years of age, who quite clearly had some neoplasm

of the orbit. She was suffering severely with pain, and I advised enucleation, with the result that there was a sarcoma of the choroid. She had no other trouble. There was no malignant history, nor any subsequent history of malignant difficulty. Recently, four years after operation, she is entirely free from any return of the growth.

At the time I reported this case I reported another which was remarkable, so far as my experience goes, in a new-born infant. I was sent for with the statement that this child had some eye trouble, and when I arrived at the hospital, about two hours after the birth of the child, there was, I found, absolute exophthalmos. The eye was literally on the cheek. I supposed that the condition was due to some accident in birth. I was unable to restore the eye to its proper position, and found that the orbit was occupied by a tumor. On examination, we found 60 different growths on the body of that child. It lived four or five days. This is the only case of that kind that I know of at that age.

The interesting point, in the case that I first spoke of, is that, after four years, there was no return of the growth.

A CASE OF PRIMARY INTRADURAL TUMOR OF THE OPTIC NERVE.

CHARLES R. HEED, M.D.,

Philadelphia, Pa.

Olive R., aged eleven years, was brought to me by the family physician, Dr. T. S. Irwin, April 13, 1915.

Family history: Both parents, a sister, and a brother living and in good health. No history of lues, tuberculosis, cancer, or cardiovascular disease in the family.

The child was born with normal labor, and until the present illness had always enjoyed excellent health. She had even escaped the usual diseases of childhood. No history of a fall or blow upon the head or about the eyes was obtainable. She attended school regularly and was well advanced in her studies.

In April, 1914, shortly after being vaccinated, the mother noticed that the left eye appeared more prominent than the right. The course of the vaccination assumed a rather marked reaction, such as swelling and discoloration of the arm and a low-grade fever. The exophthalmos slowly increased. In August, four months after the mother had first noticed the condition, the eye was very prominent and vision reduced to the perception of light. The following month the patient was taken to a surgeon, who ordered a course of mercury and the iodids, and these were continued for a period of six months with no apparent effect, the protrusion of the eyeball slowly increasing. During the entire period, or from the beginning of the proptosis, there was an entire absence of headache, pain in the eye, or vertigo.

Ocular Examination.—Right eye, vision $\frac{6}{5}$; anterior ocular segment healthy; pupil 5 mm., responded freely; rotations were normal. Eye-ground was healthy; disc margins were well defined; central cupping noted, and retinal vessels not distended. Hyperopia 0.5 D. Left eye, no light perception; eyeball proptosed approximately 2 cm., the summit of cornea directed down and in. Rotations downward and inward only slightly restricted; upward absent; externally, decidedly limited. Pupil 4.5 mm. and fixed (posterior synechia); tension normal. There was no pulsation nor bruit, and palpation failed to outline any growth in the orbit. Pressure over the closed lids did not lessen the proptosis. The iris was fixed to the lens-capsule by synechia, otherwise the media were clear. Optic disc grayish white, with margins blurred and irregular. Arteries contracted; the veins full and tortuous. There were numerous small white areas of degeneration throughout the fundus, and a large, star-shaped formation in the macula.

Enucleation of the eye had been recommended by the family physician and the parents had consented. Exploration of the orbit, with the possibility of exenteration, was considered advisable. Both the physician and parents being anxious for immediate relief for the child, arrangements were made for operation the same afternoon. An opportunity was, therefore, not afforded of securing a photograph,

Fig. 1.—Appearance of eyeball and tumor.

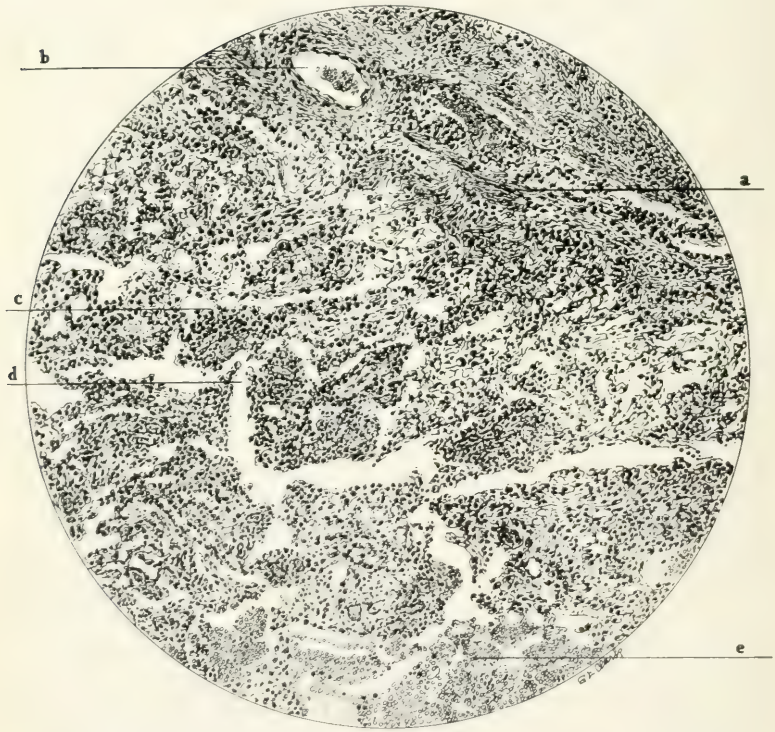


Fig. 2.—Primary intradural tumor of the optic nerve. *a*, Optic nerve tissue; *b*, blood-vessel; *c*, tumor-cells; *d*, intracellular space; *e*, large blood-space.

or having a thorough examination of the sinuses, or the making of stereoscopic radiographic plates.

Under ether anesthesia an external canthotomy was performed, the conjunctiva incised toward the ball in order to make a preliminary exploration of the orbital cavity. Dr. W. M. Sweet, whom I consulted and who kindly offered his assistance at the operation, readily diagnosed an intradural tumor, and advised against exenteration, which I had previously considered. The tumor extended from the globe well back to the optic foramen. The nature of the tumor was recognized, but its great size, and the certainty that with the displacement of all the tissues of the orbit an attempt to save the eyeball would have resulted in an enophthalmos, it was decided to enucleate with the tumor mass. So completely did the tumor fill the orbit that it was with difficulty that the finger could be passed between it and the walls of the orbit. The nerve was cut close to the optic foramen, leaving the tumor attached to the globe, as shown in the drawing (Fig. 1) and in the gross specimen, both of which were prepared in the laboratories of the Jefferson Hospital.

Pathologic Examination.—The tumor is an irregular, spindle-shaped mass, of a dark-red color, and measuring 35 mm. in length and 26 mm. at its point of greatest thickness. The capsule of the tumor is a stretched band of fibrous tissue—the dural sheath. This membrane is not involved except for a short distance adjacent to the sclera, where it appears to be slightly adherent.

The study of the slides, as reported by Dr. E. D. Funk, is as follows:

The tumor consists essentially of a richly cellular, somewhat fibrillated material, in which are numerous blood-vessels and large, irregular spaces without definite limiting walls. The cells are both oval and round in shape, and possess nuclei that vary in size, some corresponding to the size and staining reaction of the nuclei of the small round-cell or lymphoid cell found so frequently in tissues. Others are larger, conforming more to the type of the large lympho-

cyte, and stain less deeply. The cytoplasm of most of the cells is indistinct. The cells are, for the most part, rather closely placed, lying in a network of fine fibrillated material. These fibrils in some places apparently form the cell outlines, while in other instances they inclose empty spaces. These latter are probably areas of pericellular edema, but in some places are very suggestive of adipose tissue (Fig. 2). Unfortunately, the tissue was not prepared for the specific neurogliar stain.

The blood-vessels vary considerably in size, and, in many instances, have thick, fibrous, or partly hyaline walls, lined by flattened endothelium. Some of the irregular spaces previously mentioned are partly filled with red cells and appear to be blood-channels, although they are bounded by condensed tumor tissue instead of definite walls. Others of these spaces are empty, probably due to edema. At points are also many small hemorrhages into the tumor. The growth is intimately connected with the pial sheath; whether this is the point of origin is difficult to decide, but the appearance lends weight to this view. The nerve is edematous, hyperemic, and the interstitial tissue slightly more cellular than usual.

Dr. W. M. L. Coplin, who examined the sections, believed that the tumor in this case might properly be classified as an endothelioma. He also suggested that the condition found was analogous to a lesion involving the cerebral surface, spoken of as internal hemorrhagic pachymeningitis.

The subject of intradural tumors of the optic nerve has been exhaustively studied by Byers, Parsons, and, more recently, by Hudson, so that the essential features of these cases are generally known. The present case agrees in history, symptoms, and general characteristics to the majority of those already reported—early age of patient, entire absence from pain, no trauma, slow development of the proptosis, general good health of the patient, the presence of a postneuritic atrophy, and the inability to palpate any orbital growth.

The specimen would indicate a possible intracranial ex-

tension, but after removal of the tumor there was no palpable evidence of any growth at the foramen.

The child has been in excellent health since the operation, has a perfectly good socket, and wears an artificial eye with comfort.

A PRIMARY TUMOR OF THE OPTIC NERVE SUCCESSFULLY REMOVED, WITH PRESERVATION OF THE EYEBALL, BY THE KRÖNLEIN METHOD.

ARNOLD KNAPP, M.D.,
New York City.

M. J., aged nine, was brought to the hospital in December, 1914, because of a prominence of the right eye which had been noted for one year and which was slowly increasing. On examination the right eye is pushed straight forward in the axis of the orbit (Fig. 1), the motility of the eyeball is normal, palpation reveals no tumor in the orbit, sight is reduced to perception of light in the extreme temporal field. Ophthalmoscopically the disc shows a neuritic atrophy. Examination of the nose is negative. The diagnosis of tumor of the optic nerve is made and an operation advised.

Operation.—December 31, 1914. Under ether the usual incision in the Krönlein operation along the outer margin of the orbit is made. The bone-flap is formed with osteotome and mallet and reflected. A horizontal incision is made in the orbital periosteum, and the small finger is carefully introduced. This encounters an elongated swelling continuous with the optic nerve, which extends from the eyeball to the apex of the orbit. The surface of the swelling is smooth and soft. Between the tumor and the eyeball there is a small portion of apparently normal optic nerve. Deep retractors are introduced, and the mass is exposed. It is found to be bluish in color. With the finger the tumor is isolated from the surrounding tissues and the optic nerve divided directly behind the eyeball. This part of the tumor

is then grasped with a forceps and the rest of the mass freed and cut through as near the apex of the orbit as possible. During this manipulation a good deal of soft, jelly-like tissue is expelled through the cut end of the tumor. There was some hemorrhage. The flap is replaced, deep catgut sutures are introduced, and the skin incision is united with silk. At the close of the operation slight exophthalmos was still present. As this was believed to be due to hemorrhage, the eyelids were sutured together.

During the next twenty-four hours the patient vomited a number of times. The upper lid shows some blood staining. A wet dressing is applied.

January 4th: The swelling has not increased, the skin of the lid is soft, there is no fever, and the patient feels well.

January 5th: The temperature rose to 102° F. The boy complains of some headache. The region about the incision is somewhat swollen. The sutures are removed and the subcutaneous tissue opened into. This was followed by the escape of considerable blood and pus.

January 15th: The infection, which seemed to have been localized to the subcutaneous tissue, rapidly subsided and the patient is now able to open his eye. The cornea is anesthetic; the motility of the eye is good, except to the outer side; the pupil is moderately dilated; the optic nerve is white. Retinal vessels as they emerge from the disc are absolutely normal in size. There is no pulsation on pressure of the eyeball. In the region of the macula, or perhaps a little beyond the macula, there is a whitish area which does not look like edema, but rather more like a deeply situated retinal exudate.

January 25th: The wound is closed; there is still slight swelling of the eyelids; the position of the eye is good; some of the vessels of the retina are distinctly contracted; the whitish area in the macula has disappeared, leaving a few spots. The fundus is otherwise normal.

February 20th: The motility outward has improved, the pupil is dilated, the vessels are smaller to-day than they have previously been. The optic nerve shows a further stage of neuritic atrophy, and the whitish changes in the macula look like connective-tissue changes with some pigmentary deposits in the underlying choroid.

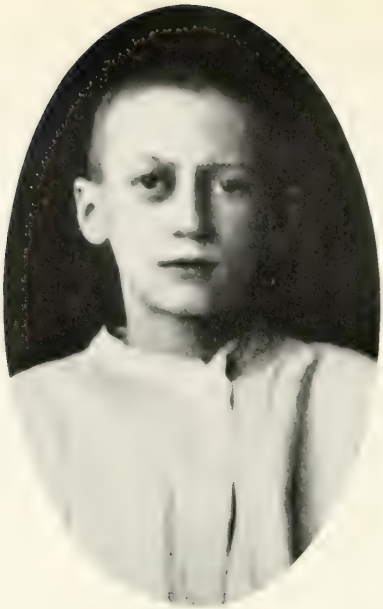


Fig. 1.—Before operation.

Fig. 2.—After operation.

Fig. 3.—Showing the extent of lateral motility after operation.

March 10th: The pupil is dilated; motility outward is better; the inferior blood-vessels are well filled.

June 22d: The position of the eye is normal (Fig. 2). There is no swelling of the lids; except for a dilated pupil, the eye looks perfectly normal; motility outward is completely restored (Fig. 3). The retinal vessels are partly normal in size, while some are extremely narrow to the point of obliteration, where they are transformed to white lines. The region of the macula is occupied by an area composed of pigment and atrophy. On the nasal side of the disc there are some areas of slight choroidal atrophy and connective-tissue changes in the retina. The optic nerve is completely white. The scar of the incision is a linear one and not noticeable.

The excised part of the optic nerve and tumor consists of an irregular, cylindric-shaped mass 3 cm. long by 2 cm. wide at its broadest part. The mass is entirely surrounded by a connective-tissue capsule which is the extended dural sheath. Within this sheath, at the cut extremities of the tumor, a smooth bluish tissue can be seen which is the capsule of the tumor proper. This is presumably the pia. Within this pial sheath there is a swollen, edematous, bluish-red-looking tissue, with a central defect which at the part of the tumor nearest the apex of the orbit occupies nearly the entire tumor. The tumor is hardened in formol and in Müller's fluid. Paraffin and celloidin sections are made.

Microscopic Appearances.—In the sections of the tumor stained with hematoxylin-eosin, nearest to the eyeball the general structure still resembles the optic nerve. The interseptal spaces are distended, the septa are irregularly hypertrophied, and there is a large increase of the glial elements. This proliferation of the glial tissue is particularly marked along the margin of the spaces, where it is continuous with tissue in the sheath (Fig. 4). The cells have round or oval nuclei, with irregular and indefinite protoplasmic bodies situated in a branching network of fibrillæ. The Weigert-stained specimens show irregular and degenerated nerve-fibers. Toward the center of the nerve the interseptal spaces become empty, and only the connective-tissue trabeculæ with their vessels are present in between a blue-stained amorphous tissue. The blood-

vessels are dilated but do not show any change in their walls. The dura is normal. The pia is irregularly thickened by an infiltration of tissue whose cells vary in different parts. In one place where the central vessels enter the optic nerve the glia-cells are directly continuous with the cells in the pia, where they are crowded together. In other parts the pia is greatly thickened by these invading cells, which are often separated by large clefts in the tissue filled with a blue material.

Proceeding backward from the eyeball, the changes within the neural portion of the optic nerve become more marked, and the configuration of the optic nerve is entirely lost. Within the widely distended pial and dural sheaths there is a large cavity which, before operation, contained the jelly-like material expressed during the operation. The margin of this artificial cavity is formed by proliferating tissue springing from the pia, which is curiously lobulated (see Fig. 5). Under the high power this tissue is very cellular, the nuclei are round or oval, the cell-bodies cannot be identified except for an occasional fusiform or branching tendency; they are separated by vacuoles and cystic spaces, and replaced by a substance which stains blue. The great vascularity of this tissue is very striking and suggests a hemangioma; the vessel-walls show no degenerative changes.

Under "Tumors Arising in Tissues Derived from the Mesoblast," Collins and Mayou* describe the new-growths of the optic nerve as follows: These are most frequently neurofibromata. In some tumors there is an overgrowth of all the connective-tissue constituents—a general fibromatosis. In others, some parts are exclusively affected. Thus the growth may be entirely in the nerve itself, starting from the neuroglia and fibrous trabeculæ, without involving the sheaths. Not uncommonly the pial sheath and the fibrous tissue are affected and the dura escapes. The new-growth is composed of fibrous tissue, either fully formed or of an embryonic type. Cystic spaces containing fluid may form

* Collins and Mayou: Pathology and Bacteriology of the Eye.

Fig. 4.—Section from tumor near eyeball, showing neuroglia proliferation directly continuous with the infiltration of the pia and of the sheath.

Fig. 5.—Section from tumor near apex of the orbit, showing the lobular condition of the tumor springing from the internal surface of the pia, the central cavity formerly containing a myxomatous-like tissue.

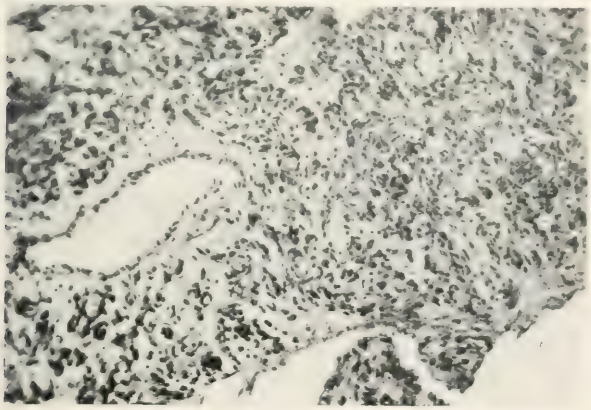


Fig. 6.—High power of this portion of the tumor.

within the growth. The fluid has not been found to contain mucin, and there does not appear to be any real evidence of myxomatous degeneration.

Parsons* regards these tumors as belonging to the endotheliomata, and believes that the neuroglial proliferation is a subsidiary phenomenon.

A. C. Hudson† gives an exhaustive review of 118 primary tumors of the optic nerve from their clinical and pathologic sides. He agrees with Fischer in designating those tumors as examples of gliomatous degeneration, and suggests the following modification: "Degenerative gliomatosis implying a generalized overgrowth of neuroglial tissue of infiltrative character dependent upon some degenerative change in the tissues of unknown origin."

Our tumor shows a general neuroglial proliferation, with myxomatous-like degeneration, though the main seat of the process seems to be in the pia. A transition of the two parts of the tumor can be made out. The specific neuroglia stain unfortunately did not succeed, owing to improper fixation of the specimen.

The clinical features of this case do not differ from those generally found in this well-defined group of tumors.

The operative procedure, the Krönlein osteoplastic resection of the outer bony wall of the orbit, proved perfectly satisfactory, as it furnished free access to the site of the tumor and permitted the preservation of a normally appearing eye without any subsequent disturbance of motility.

An interesting feature in the course after removal of these optic-nerve tumors is the changes in the retinal circulation which occur. In our case it was very remarkable that, notwithstanding complete division of the optic nerve and its blood-vessels, the retinal circulation apparently was unaffected, the vessels being for at least three weeks of normal

* Parsons: Pathology of the Eye.

† Hudson: *Ophthalmic Hospital Reports*, 1910, vol. xviii.

size, both the arteries and the veins, except that pressure exerted upon the eyeball elicited no pulsation. Subsequently some of the vessels became narrow and obliterated. The inferior temporal vessels, however, are at the present date still of perfectly normal size.

Schlodtmann,* in his monograph on the "Extirpation of Retrobulbar Tumors," has given this feature particular attention, and shows that the changes in the ophthalmoscopic picture of the retinal circulation after operation depend upon whether a collateral circulation has been established or not. It is evident that a tumor obliterating the optic nerve will interfere with the central retinal vessels. Under these circumstances a collateral circulation from the posterior ciliary blood-vessels and the retina must take place, and, as the latter vessels may partly or completely escape obliteration through the operation, the subsequent circulation of the retina will be carried on by these vessels. If this collateral circulation has been developed, no evidences of an arrest of the retinal circulation (ischemia, edema of the retina) occur. The pigmentation and atrophies which are observed in the eye-ground are presumably the evidences of a disordered circulation.

A point to which I want to draw particular emphasis is the incomplete removal of these optic-nerve tumors, which, according to Hudson, occurs in 50 per cent. Thus, in the case just described, the tumor process was not limited to the apex of the orbit, and unquestionably has extended into the cranial portion of the optic nerve. At present there are no signs, however, of any intracranial extension. In the cases on record not a single instance of local recurrence in the orbit has been noted.

These tumors are generally regarded as almost benign. It seems to me that Byers is correct in warning against this assumption, even if it is years before the intracranial exten-

* Schlodtmann: Vossius' *Sammlung zwangloser Abhandlungen,* 1900.

sion shows itself. As the *x*-ray seems to possess a distinct effect on the growth of gliomatous tissue,* should not systematic treatment with the *x*-ray constitute a part of the treatment of these cases?

A CONTRIBUTION TO THE SUBJECT OF TUMORS OF THE EYELID AND ORBIT.

G. E. DE SCHWEINITZ, M.D.,
Philadelphia, Pa.

CASE I.—*Myxosarcoma of Right Eyelid of Unusual Size.*†—The case history of the patient from whom this growth was removed postmortem is necessarily brief and incomplete, but the essential facts can be detailed.

A boy, aged four, came for examination on July 24, 1914, to Dr. J. Chalmers Da Costa, who very kindly afforded me the opportunity of studying the condition with him.

There is nothing of importance in the family or in the patient's history which relates to the disease from which he suffered, except as follows: In June, 1914, slight ptosis of the lid was noted, and the child was taken to Dr. Haskin, of Williamsport, by his physician, Dr. Poust. Dr. Haskin reports as follows: "Upon examination I found a small growth on the inside of the upper right eyelid, resembling an ordinary hematoma, which had been discovered only a day or two before, although it had been noticed that this eye was not quite natural for two or three weeks prior to my examination. I gave the child an anesthetic, opened the growth, thoroughly cureted the diseased area, and packed it with iodoform gauze. The contents of the growth had the appearance of granulation tissue, but no microscopic examina-

* Axenfeld: *Klin. Monats. f. Augenheilk.*, 1915, No. 1.

† This tumor was exhibited at a meeting of the Pathological Society of Philadelphia by Dr. Shumway and myself, but its description was not published.

tion was made at that time. Six days later the child returned with the tumor in evidence and much increased in size. Again it was excised, the base thoroughly cureted, cauterized with the actual cautery, and the cavity packed with iodoform gauze. The patient remained in the hospital for a few days, but even before he left the ward the growth had begun to recur. The tissue removed at this operation was carefully investigated by Dr. Weddigen and found to be sarcomatous."

When I examined the patient, two weeks after the first operation described by Dr. Haskin, the upper lid was greatly swollen and bluish in color. Protruding through the palpebral fissure there was a large mass of friable tissue about the size of a walnut, somewhat resembling granulation tissue, reddish yellow in color, and bleeding freely. So far as could be determined the eyeball and orbit were uninvolved. A fragment of the mass was removed and immediately examined by Dr. Allen J. Smith, who confirmed the previous diagnosis. Radical operation was advised, but the parents declined to permit it.

The child was not again seen. He died November 11, 1914, therefore four months after the first removal of the small, hematoma-like body from the eyelid described by Dr. Haskin. There was no general autopsy, but the physician, Dr. Poust, kindly removed the specimen, and in his letter says: "The mass was attached by a pedicle which grew from the under surface of the right upper lid. There was no tumor-mass on the surface of the lid. When traction was made upon this growth the eyelid moved with it, and its origin was in the upper culdesac" (Fig. 1). The weight of the tumor was 247 grams; its size was 10 by 8 by 6 cm.

Sections from various portions of the tumor-mass are identical in appearance. They exhibit a scattered and scant trabecular framework of fibrous tissue, usually bearing the major vessels, which are visible and not sharply defined from the rest of the tumor tissue. The latter in places is composed of practically pure myxomatous connective tissue (a delicate reticulum of small stellate cells); in other places of pure, small, spindle-cell sarcoma and of muscle, with more or less admixture of both types of tissue, the sarcomatous tissue being sharply defined from the gelatinous connective tissue,

Fig. 1.—Case I. Myxosarcoma of orbit. Weight, 247 grams; size, 10 x 8 x 6 cm.

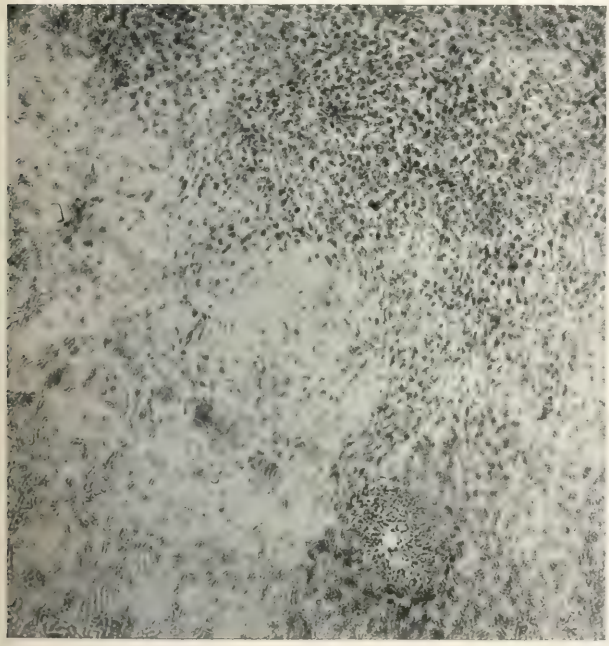


Fig. 2.—Case I. Photomicrograph. Myxosarcoma of orbit.

Fig. 3.—Case I. Round-cell sarcoma of eyelid.

and sometimes slowly grading into it. Blood-vessels of typical sarcoma type, that is, mere endothelial channels in the midst of the cells, are fairly numerous in the midst of the fields. Vessels in the myxomatous areas are small and sparsely scattered (Fig. 2).

Comment.—While it is usually stated that sarcomata of the eyelid are comparatively uncommon, they are probably not so rare as the number of reported cases indicate. The literature of the subject has been carefully collected by Wilmer,* who gathered 35 cases in 1894; Veasey† added 11 more in 1899, and Alling‡ brought up the total to 65 in 1907. In 1911 Dr. Shumway and myself § recorded a case of lid sarcoma. At that time we found that there were approximately 80 cases of this disease on record. This sarcoma I removed from the upper lid of a negress aged eighty-one. The tumor measured 4 cm. in length and 3.75 cm. in breadth, and hung down over the cheek. The preauricular and submaxillary glands were involved by metastasis. Under local anesthesia the mass was dissected from its position between the tarsal cartilage and the skin. Microscopically, it was a round-cell sarcoma free from pigment. Dr. Evans, the patient's physician, reports that she lived more than a year after the operation, and died from pneumonia (Fig. 3). There was no recurrence.

In examining the literature of the subject at that time Shumway and myself found that in 43 per cent. of the cases the sarcoma was round-cell; in 40 per cent. spindle-cell; while in the remainder the cellular type was mixed. Parsons believes that some of the so-called round-cell sarcomata of the lid should be considered as lymphomata. Between 30 and 40 per cent. of these lid tumors are pigmented. There

* Wilmer: *Trans. Amer. Ophth. Soc.*, vol. vii, 1894–1896, p. 91.

† Veasey: *Trans. Amer. Ophth. Soc.*, vol. viii, 1897–1899, p. 519.

‡ Alling: *Ophthalmic Record*, June, 1907.

§ *Trans. Section on Ophthalmology, College of Physicians of Philadelphia*, February 16, 1911.

appears to be some difference of opinion in regard to the frequency of myxosarcoma in these circumstances. Michel,* for example, refers to only one case of this nature, which was reported by Van Duyse and Cruyl in 1887, while Parsons records 11 per cent. of myxosarcomata. The tumor referred to by Michel occurred in a seven-year-old girl, and is said to have followed a blow upon the eyelid and to have grown very rapidly. At the time of operation the growth had already exceeded in size a child's fist. Microscopically, the structure was that of a myxosarcoma with small spindle-cells, their origin being in the loose cellular tissue between the tarsus and orbicularis. In other words, this growth almost exactly resembled the one which I now put upon record. Plexiform angiosarcomata and cylindromata are said to occur more frequently than myxosarcomata.

In Wilmer's list the age of the patients affected varied between ten months and seventy-six years. As already noted, I have removed one sarcoma from the lid of a patient who was eighty-one years old. The size of the growth has varied from that of a pea to that of a large apple. The growth which forms the subject of this brief report very much resembled in appearance the cerebellum. From the lids these tumors may spread to the orbit or to neighboring tissues, and even when thoroughly removed, are prone to recur, sometimes with startling rapidity, as in the present instance.

CASE II.—*Small Round-cell Sarcoma of the Orbit.*—The patient from whom this growth was removed is a married woman, aged forty-nine, who came to the University Hospital in July, 1914, because of a marked proptosis of the left eyeball and all the evidences of a highly vascular orbital growth. At that time the appearances were as follows: The globe was depressed 2 cm. below the level of the right eye, and the face of the cornea turned somewhat inward. The

* Michel: Graefe-Saemisch Handbuch der gesammten Augenheilk., 2d edition, 1908.

Fig. 4.—Case II. Front view. Sarcoma of orbit.

Fig. 5. -Case II. Side view. Sarcoma of orbit

Fig. 6.—Case II. Sarcoma of orbit and exposure keratitis. Condition just prior to operation. Compare with Fig. 4.

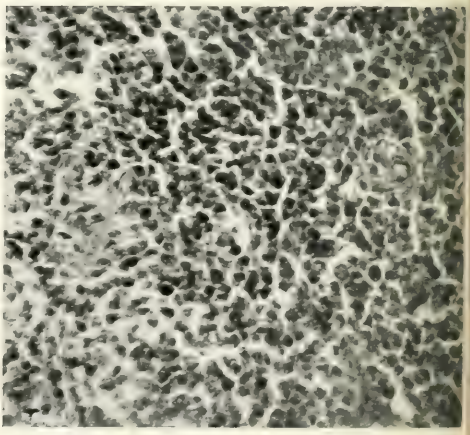


Fig. 7.—Case II. Photomicrograph. Sma round-cell sarcoma of the orbit.

overlying lid was swollen and stretched, and its veins were greatly enlarged. The upper bulbar conjunctiva was engorged and covered with enormously dilated veins. The upper part of the orbit contained a large mass of tissue, which felt like a congeries of vessels, and was somewhat elastic to the touch. Upward and outward rotation was lost; downward and inward diminished ocular movement was present, but if pulsation was present, it was extremely slight; there was no thrill and no bruit of which the patient was conscious. Although the cornea was roughened, the fundus could be studied. The disc was pale; no swelling or haziness of its margins; the retinal veins were large. V. = $\frac{6}{45}$. Hertel's instrument registered 34 mm. The functions of the right eye were normal in all respects, and in the fundus no abnormalities were detected; uncorrected vision = $\frac{6}{15}$; refraction, M. (Figs. 4 and 5).

The patient positively declined any operative interference, and returned to her home in the country. She was readmitted on January 27, 1915, when the following history was obtained: Five years prior to this period the first symptom noted by the patient was a downward deviation of the left eye, together with diplopia. Gradually there occurred increasing protrusion and prominence of the eyeball. For four years the growth was slow in development, and only during the last year of its existence was there any impairment in vision. During the fifth year of its existence the tumor increased rapidly in size, and was associated with marked swelling of both eyelids and ever-increasing redness, together with injection of the palpebral veins. Sight failed rapidly during the three months prior to her readmission to the hospital, and one week before she came for this examination purulent keratitis developed. No pain had been experienced at any time in the eyeball, the orbit, or the head.

The patient's past medical history is negative in so far as it has any bearing upon the growth in question; so, also, is the family history negative. Examination revealed a well-built woman, without any glandular enlargements, normal heart and lungs, no renal affection. Investigation of the abdomen yielded negative results, and, in point of fact, there were no abnormalities on a very thorough general examination. The left eye was markedly proptosed and pushed

downward, so that it protruded between the swollen lids, hung down upon the cheek, and was surrounded by a thick, vascular, chemotic conjunctiva. The lids themselves were enormously swollen, of a reddish-purple hue, and over them coursed thick, purplish, injected veins. A great mass of these veins, almost the size of fish-worms, covered the upper conjunctival region. There was indistinct pulsation on palpation. The eye was entirely blind, with a large crescentic ulcer just below the pupil space, and a thick hypopyon (Fig. 6). The right eye was normal in all respects.

Operation.—This was performed on January 28, 1915, and as the external character of the growth suggested the possibility of severe hemorrhage, preparations were made by Dr. George Müller for prompt ligation of the carotid in case this became necessary. The eyeball was first enucleated and found to be unconnected with the tumor, and with the finger a rather soft, apparently highly vascular, tumor-mass could be felt filling the orbit. Complete evisceration of the contents of the orbit was effected, and the orbital periosteum stripped from the bones. These were apparently uninvolved, except at one suspicious spot on the inner side, which had been covered with periosteum darker in color than elsewhere, and which was situated directly over the ethmoid plate. Hemorrhage was readily controlled by packing, and the lid margins were pared and stitched together with silk, leaving a small opening for drainage at the outer canthus. The patient showed no ill effects from the operation, and there was no redness nor swelling of the surrounding tissues. The packing was removed at the end of forty-eight hours. For a day or two there was a moderate, semipurulent discharge, quickly controlled by daily douching of the cavity, first with saturated boric-acid solution and later with permanganate irrigations. Convalescence was prompt, and the patient left the hospital eighteen days after the operation.

The patient's physician, Dr. J. C. Atkins, who had kindly referred her to me for operation, reports on June 7, 1915, that six months after operation there is no recurrence, and, except in one small spot, the orbit is covered by integument. The patient is in excellent general condition.

Sections were prepared from various portions of the tumor mass, and studied by Dr. Allen J. Smith and myself. The

tumor-mass was of soft, fleshy consistence, and in bulk large enough to fill an ordinary drinking glass. The sections of the growth demonstrate that it is a small round-cell sarcoma, infiltrating the loose fibrous tissue of the orbit, from which the fat, so far as sections thus far examined give evidence, has been absorbed. Sarcomatous tissue infiltrates the muscles and enveloping muscle-fibers, previously existing vessels and nerves, the tumor-cells being of the ordinary small lymphoid type, with large, highly chromatic nuclei and small cytoplasm. There is little intercellular substance save where the infiltrated tissue provides an intercellular matrix. The growth is very rich in blood-vessels, many of them being of the channel type common to sarcomata, but there are also many vessels with definite walls. These vessels probably belong rather to the original tissue than to the neoplasm. The sarcoma cells can be traced throughout the trabecular structure of the lacrimal gland, but the glandular cells themselves do not appear to be diseased. Sections of the eye indicate a beginning invasion of the tumor-cells into the sclera in the anterior portion of the bulbus. There is a marked purulent keratitis and hypopyon. The iris is acutely inflamed, and there are inflammatory edema and hyperemia of the ciliary region, and a similar change throughout the vascular layers of the choroid, most marked in the anterior half of the bulbus. All the vessels of the choroid and sclera are markedly engorged. The retina is detached. There is well-marked papilledema of the optic nerve, and some interfascicular lymphocytic infiltration (Fig. 7).

CASE III.—*Additional History of an Intradural Tumor (Psammo-endothelioma) of the Optic Nerve.*—The first communication on this growth was presented to the Section on Ophthalmology of the College of Physicians;* in order to complete the history of the patient, I quote from the paper referred to as follows:

Mary S., aged eleven, came to the University Hospital Eye Dispensary on June 30, 1910, on account of a slowly increas-

* *Trans. Section on Ophthalmology, College of Physicians of Philadelphia.* The paper is here briefly summarized.

ing right exophthalmos which had been evident for some years. The family and personal histories are unimportant in that they have no bearing upon the origin of the intradural tumor, save only in one respect, presently to be described. Physical examination of the girl demonstrated that she was well nourished; there were no signs of general adenopathy. The chest was normal in appearance, except for a prominence of the lower part of the sternum. Her heart, lungs, and abdominal organs were in their correct positions, and there were no external appearances of abnormality. In each iliac fossa there was an area of tenderness. The urine contained a trace of albumin, otherwise it was normal. The blood examination yielded the following results:

Erythrocytes	4,440,000
Leukocytes	7,200
Hemoglobin	87 per cent.
Polynuclears	71
Lymphocytes	21
Mononuclears	2
Eosinophils	0

Right Eye.—V. = no l. p. Hertel's exophthalmometer registered 17 mm. The eye was divergent and slightly depressed; the lateral movements were unimpaired; the upward and upward and outward rotation was limited. The media were clear, the disc round, entirely atrophic, but there was no shrinking of the vessels, and there were no signs of a preceding neuritis. The pupil failed to respond to light, and the consensual reflex to the left eye was not transmitted. H. = 3 D.

Left Eye.—V. uncorrected = $\frac{6}{12}$; later correction (H.As.) secured normal sight. There were no ophthalmoscopic evidences of disease.

The defective vision of the right eye has been present since the patient was four years of age, and is said to have arisen after the child was greatly shocked by a lightning bolt, which struck the porch on which she was sitting. She was thrown from her seat, but it is unknown whether there was any period of unconsciousness. Associated with the loss of vision the exophthalmos gradually developed, but has been exceedingly slow in its elaboration.

She was admitted to the hospital, and careful general and

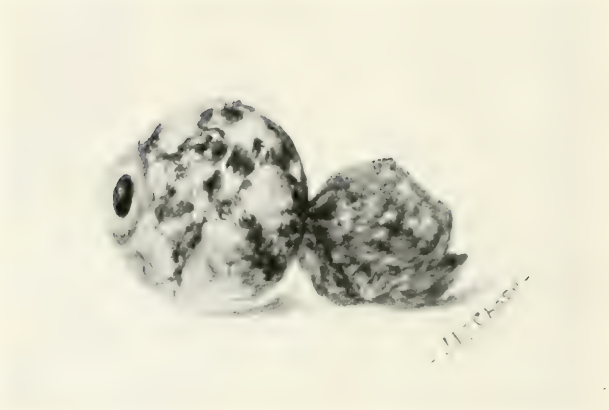


Fig. 8.—Case III. Psammo-endothelioma of optic nerve.

local studies were made, which, except for the evident orbital lesion, or, at least, suspected lesion, were negative. *X*-ray examination failed to reveal any change in the orbit or any abnormalities of the accessory sinuses. The symptoms were best explained by assuming the presence of an optic-nerve tumor. Operation was advised but declined.

Four months later she returned, and the conditions previously described remained unchanged: the exophthalmometer registered 17 mm. O. D., 13 mm. O. S.

The patient reported from time to time, but no change in the exophthalmos was discovered until early in the spring of 1914, or four years after the first examination and about eight years after the first signs of proptosis had appeared, when the patient began to experience deep-seated orbital and periorbital pain, and an increase in exophthalmos was evident, Hertel's instrument registering 20 mm. In addition to this, attacks of sharp pain at the angle of the orbit were frequent, as well as pain in the temporal and parietal region; she also suffered frequently from occipital headaches. She believed that the vision of the left eye was failing, but the ophthalmoscope failed to reveal any lesions, and the central and peripheral vision were normal.

Operation was now urged, and consent for its performance was given. The external rectus was severed and drawn outward with a thread. With a protected finger it was easily ascertained that the posterior part of the orbital cavity was occupied by a dense growth of oval shape, proceeding from the posterior surface of the globe to the apex of the orbit. It was intensely hard to the touch and firmly attached to the eyeball and to the surrounding tissue. It was impossible to remove it without sacrificing the eyeball, which, therefore, was enucleated with the attached growth, the latter being gradually separated with the fingers and an Allis dissector and ultimately removed. Its appearances are depicted in Fig. 8. The tumor was 2.5 cm. in length and 2 cm. in width, dense in structure, and reddish yellow in color.

The patient's convalescence was comparatively uneventful. For a period of time there was complete ptosis. Gradually the levator regained its power, and by the beginning of July, or three months after the operation, the ptosis had entirely disappeared and an artificial eye was adjusted. About

one month later the patient had some return of periorbital pain and there was a suggestive fulness in the orbit, especially upward and inward. This increased, and at the expiration of another month there seemed no doubt that a recurrence of the growth was taking place.

On September 24, 1914, the patient could no longer wear the artificial eye, and she was admitted to the hospital and radium applied for eight hours by cross-fire outside of the orbit. This application was twice repeated, one application being within the orbit, with the result of distinctly lessening the swelling and decidedly decreasing the pain.

Sections prepared from this growth present the histologic picture of a psammomatous endothelioma. The bulk of the mass is made up of a coarsely and irregularly interwoven network of fibrous tissue, in the midst of which are found numerous concentrically laminated psammoma bodies. In parts of the growth are groups of comparatively large embryonic connective-tissue cells of variable shape, provided with fairly large, round, and oval nuclei of vesicular type. These are apparently of endothelial origin, and in picked fields their relation with small blood-vessels is clear, such vessels being seen in longitudinal section, often with branches, showing their walls formed of multiple layers of these elements; or they are visible in transverse section as whorls of endothelial cells, sometimes with the diminished lumen still appreciable, sometimes solidly closed. In these more or less fusion of the cells may be made out, apparently with hyaloid change; and in a few places the development of the psammoma bodies in these masses of endothelial proliferation is evident. (See photomicrographs prepared by Dr. Allen J. Smith, Figs. 9 and 10.)

For a time we were encouraged to believe that radium treatment would prove to be effectual, but in spite of it there was a gradual development of the recurrent growth, and toward the end of January, 1915, the indications were pressing for complete evisceration of the orbital contents. This operation was performed on January 28, 1915, and much difficulty was experienced in removing the mass, almost cartilaginous in its density. Ultimately the plate-like masses were displaced, together with the periosteum, with which they were intimately connected. The orbital cavity was

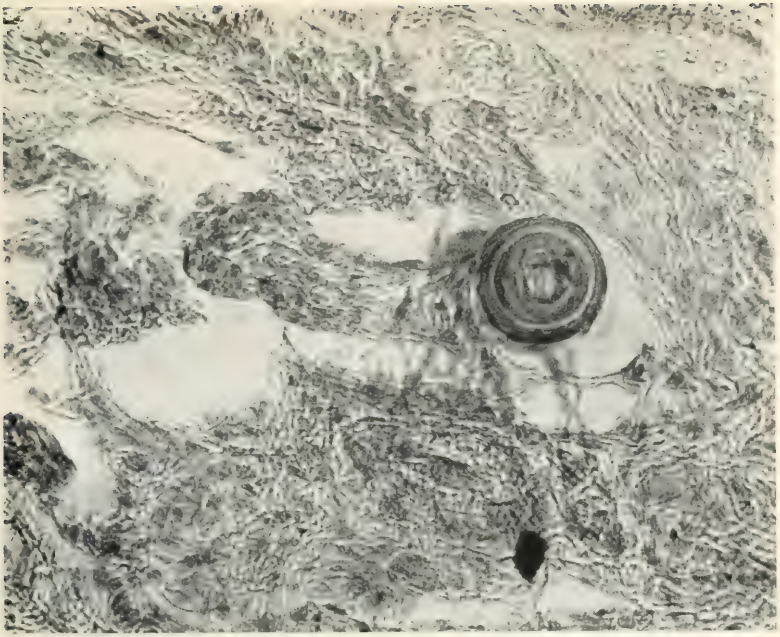


Fig. 9.—Case III. Psammo-endothelioma of optic nerve. Photomicrograph showing formation of a psammoma body in a small blood-vessel.

Fig. 10.—Case III. Psammo-endothelioma of the optic nerve. Photomicrograph showing development of psammoma bodies in small blood-vessels.

lightly packed with gauze, which was removed at the end of forty-eight hours. The subsequent treatment consisted in daily irrigations of potassium permanganate solution (1:5000) until healing was complete.

Microscopic examination of sections from the tissue demonstrates that it is mainly composed of dense scar material, with scattered inclusions of adipose tissue, but infiltrating roots of spindle-shape and round connective-tissue cells of ordinary size and type are also visible.

As before, psammoma bodies are present in the cellular tissue, and a few are visible in the dense scar tissue. The same relation of these concretions with blood-vessels as this was observed in the original growth is again easily recognized. The cells of the recurrent tumor are less frankly endothelial in type, in comparison with those found in the original neoplasm, and suggest an ordinary small polymorphous sarcoma; but in places, especially about the bodies of the cellular roots, cells of larger and more definitely endothelial character may be seen. The fibrous matrix into which the tumor is infiltrating is doubtless the original orbital tissue, increased and indurated by cicatrization following the first operation.

Referring now to this intradural tumor of the optic nerve, the following features of the report may be emphasized:

(*a*) The possible relation of injury to this growth and its development; such relationship is always vague, unsatisfactory, and perhaps unscientific, and yet it has been noted more than once.

(*b*) The long period of the quiescence of this growth, certainly four or five years, probably longer, followed by a rather rapid increase in the pressure manifestations and corresponding increase in the exophthalmos.

(*c*) The opportunity of studying the psammoma bodies. Sand bodies in these dural growths have been noted in several instances, for example, by Ribbert, and many years ago by Billroth. The origin of these bodies from vascular endothelium is clearly demonstrated, exactly as it has been described

by Adami.* According to this authority, these psammomata are practically always of endothelial origin, and he explains their appearance "as due to localized overgrowth of the capillary endothelium, at first in the main concentrically, so that the capillary becomes enlarged by the deposit of layer within layer of these endothelial cells." Thus, in the hemangio-endotheliomata of the dura mater microscopic examinations will demonstrate, according to Adami, a collection of whorls of concentrically disposed cells, and sometimes a whorl may be found that has a lumen containing red corpuscles. Whorls may also be noted which exhibit hyaline changes, and frequently there is a deposition of calcareous salts in these degenerated areas, so that small, calcareous nodules are scattered through the specimen, and the name psammoma is given to the growth.

Referring to the recurrence of this growth, which was evident within two months after the first operation, in spite of the fact that the neoplasm was apparently completely removed, it may be said that if any suspicious tissue remained, it must have been deep down at the apex of the orbit. The periosteum was evidently involved in the growth and infiltrated by it, hence the plate-like, thickened masses which constituted the chief portion of the recurrent neoplasm. Like the original tumor, it was a psammo-endothelioma.

At first the indications that the radium applications were controlling this process were encouraging, but save only that they allayed the pain, their ultimate effect was entirely indifferent.

Two months after the evisceration of the orbital contents, and with no signs of recurrence, the patient was again admitted to the hospital because of frequent attacks of almost explosive vomiting, unassociated with nausea. These attacks were suggestive of intracranial involvement, but gradually they ceased. Except a moderate gastric hyperacidity,

* Adami: Principles of Pathology, vol. i, p. 823.

nothing abnormal was found. The patient's appendix had been removed during the interval between the first and the second orbital operation, but she had convalesced from this operation without complications. The patient is now, ten months after evisceration of the orbit, in excellent health, and has gained in weight, and all her functions are normal.

From the operative standpoint it may be said: A Krönlein resection of the orbit would not have exposed the growth more satisfactorily than the method pursued, nor would it, I think, have been possible to remove it and save the eyeball: the attachments to the globe and to the apex of the orbit were too intimate. If they had been less intense, the growth could just as readily have been removed without a Krönlein as with it, a point recently well made by Dr. Byers.

CASE IV.—*Epibulbar Lipoma.*—Lipomata and lipomatous dermoids are not infrequently encountered situated between the superior and the external rectus. In the present instance the chief interest which attaches to this neoplasm is that its size is somewhat greater than is usual in these circumstances.

The growth was present in the left eye of a married woman, aged forty-one, and the following is her history: At the age of three months, on the subsidence of a severe conjunctivitis, a small, yellowish-white, non-inflammatory swelling was noticed in the region of the outer canthus, partly concealed by the upper lid. This growth was treated by an irregular practitioner, but what means were used in the hope of dissipating it is not known. Apparently it gave her parents no further concern, and it was only when the patient had reached the age of sixteen that increase in the dimensions of the tumor was noticed, such increase being unassociated with pain except during recurring attacks of conjunctivitis, when the mass is said to have become red and swollen.

The patient's past medical history, family history, and social history are negative, and the general examination revealed no abnormalities. The eye itself was perfectly normal, as was also its vision. During fixation in the median line, at the outer canthus of the left eye, a yellowish-white

swelling was visible, extending about two-thirds of the way toward the cornea, and covered by conjunctiva, which was smooth, glistening, and uninflamed. On everting the lid and making even pressure just outside of the canthus the growth suddenly bulged forward from beneath the canthus and the lids, and projected at least a centimeter and reached the edge of the cornea. There was one small inferior extension which projected from the edge of the main body of the growth.

The operation consisted in making a horizontal incision over the area of greatest protrusion, and dissecting the conjunctiva from the underlying fatty tissue, precisely as skin would be dissected from an underlying growth. The tumor extended well backward and into the orbit for a considerable distance, and was with some difficulty removed; in shape it somewhat resembled a hickory nut, and was nearly 3 cm. in length and $2\frac{1}{2}$ cm. in width. There were no anomalies of healing, although for a period of time after cicatrization was complete there was some limitation in the outward movement of the eye and momentary diplopia in efforts to look to the left. Gradually these symptoms disappeared.

The histologic examination of the tissue demonstrated that it was frankly adipose. There was considerable hemorrhagic infiltration in both the investing tissue and in places in the main tissue of the tumor itself.

As is well known, tumors in this region and of this character were originally supposed always to be ordinary lipomata, but as the number of examinations increased, and especially when serial sections of the growth were made, other tissues were found—glandular tissue and the elements of the skin as they occur in ordinary dermoids. Parsons states that tumors of this nature are always congenital, but that they may not be noticed owing to their position under the upper lid, and also because, like dermoids, the impetus of their growth may occur at puberty. They have been most frequently seen in women, and this has been my own experience. All those that I have removed have occurred in the female sex, and certainly in two out of four of these growths which I have investigated microscopically it was not possible

to find anything but pure adipose tissue, or, as Parsons puts it, the occurrence of simple lipomata cannot be eliminated. A point made by Lagrange (quoted by Parsons) is that, if the tumor is a pure lipoma, the conjunctiva moves freely over it, but if it is a dermolipoma, the conjunctiva is adherent to it.

DISCUSSION OF THE PAPERS OF DRS. KNAPP, HEED, AND DE SCHWEINITZ.

DR. W. H. CARMALT, New Haven, Conn.: If I remember rightly, Dr. Knapp's father, our late honored member, reported the first case of extirpation of a tumor of the optic nerve with preservation of the eyeball and perfect mobility. That was a case of carcinoma, as this is one of glioma.

I wish to speak particularly of the apparent success of the operation from the therapeutic standpoint. It is too early yet to speak of this positively, but even in this short time it is remarkable that there should not have been a recurrence, for, as we all know, gliomata are among the most malignant of tumors. I speak of this because it must have been some time in the sixties that I removed a glioma of the retina in a child, and, with the hope of preventing a local recurrence, I took out fully an inch of the optic nerve along with the eyeball. The tumor was apparently confined to the retina, but, in order to be as sure as possible that I had removed it all, I drew upon the optic nerve, and was able to excise it back to the optic foramen, removing, as stated, fully an inch of it, but in the course of a few weeks the tumor recurred in the stump of the optic nerve and the child died, a few weeks later, from an extension into the brain, with growths through the diploë of the temple and skull. Dr. Knapp said he did not remove the whole mass; he left a portion remaining at the end of the optic nerve. I hope there may be no recurrence, but I doubt it very decidedly.

DR. G. E. DE SCHWEINITZ, Philadelphia: Seven years ago I removed an intradural tumor of the optic nerve from a boy of fourteen. The tissue was fibroneurogliar in character, and contained some ganglion-cells, or, at least, cells which resembled ganglion-cells. There has been no recurrence, local or intracranial, but, as Dr. Knapp points out, the patient is not yet safe from this complication.

HEREDITARY POSTERIOR POLAR CATARACT, WITH REPORT OF A PEDIGREE.

S. LEWIS ZIEGLER, M.D., AND J. MILTON GRISCOM, M.D.,
Philadelphia, Pa.

In September, 1909, two boys, aged ten and fourteen years respectively, were brought to the Wills Hospital by their mother for the correction of defective vision. Ophthalmoscopic examination revealed the fact that both patients were suffering from double posterior polar cataracts of a hereditary type. There were no other ocular abnormalities present, and V. O. D. = $\frac{20}{100}$ and O. S. = $\frac{20}{70}$ in the older boy, and O. D. = $\frac{8}{200}$ and O. S. = $\frac{8}{200}$ in the younger. Operation was advised, and both patients admitted to the hospital.

Upon inquiring into the ocular condition of other members of the family it was discovered that numerous relatives of these boys had defective vision, and that various surgeons had from time to time been consulted for their relief. After a thorough investigation of all these patients and a close search of various hospital records, the accompanying pedigree was worked out, the cataracts recorded having been either examined personally or their histories obtained from reliable hospital records.

The original member of this family (Dutill) migrated to the United States from France about 1810, but no record could be found relating to the condition of his eyes. The first member of the family who was known to have congenital cataracts was a son of this original settler in America. In the second generation there were nine members, of whom six had congenital cataracts. The third generation contained 31 members, of whom 10 had congenital cataracts, and the fourth generation contained 23 members, of whom 7 showed the typical lenticular condition found in this family. In every case examined and in all the hospital records reviewed the opacity was situated on the posterior capsule, and varied

from a small round dot at the posterior pole to a dense circular disc covering the central third of the posterior capsule. No other congenital ocular defects were found, and the family as a whole was apparently up to the average of those occupying a similar social position.

A study of the pedigree presented shows that there were 64 members in the four generations, of whom 24, or 37 per cent., had congenital cataracts. The relative percentage of females affected was slightly higher than that of the males—10 out of 23, or 43 per cent., in the former, and 14 out of 41, or 34 per cent., in the latter. The rule of once free always free is followed in this family, and in no case did normal patients produce affected children. It is interesting to note that, with one exception, in the third and fourth generations *just one-half* of the children of cataractous parents inherited the condition, while the remaining half was normal. Males were much more prone to transmit the defect, since 20 out of 31 children, or 64 per cent., descending from cataractous males were affected, while only 3 out of 9 children, or 33⅓ per cent., descending from cataractous mothers, had cataracts.

	Total	Affected	Percentage
Males.....	41	14	34
Females.....	23	10	43
	64	24	37

The history of the two patients originally seen at the Wills Hospital is rather suggestive of low resisting power, both mentally and physically. Samuel D. was operated on by discission of the lens of the left eye and discharged cured in about five weeks. Three months later the right eye was operated on by discission. In a few days there developed a virulent iridocyclitis with exudate, resulting in complete loss of vision and shrinkage of the globe. After his discharge from the hospital he was not seen again for about two months, when he returned with beginning sympathetic irritation in the eye first operated on. The irritating eye was immediately enucleated, but the iridocyclitis in the right eye persisted to such an extent that, six months later, the globe became shrunken, and vision was reduced to doubtful light perception. A letter from his family physician, Dr. George W.

Miller, of Norristown, Pa., stated that some time previously the boy had suffered from a gonorrheal urethritis with such severe systemic disturbance that at one time his life was despaired of. This suggested a possible metastatic origin of the iridocyclitis. The patient is now an inmate of the Pennsylvania Institute for Instruction of the Blind.

The younger brother, Schley D., had been operated on in the right eye for a similar type of cataract by another surgeon two years previously, and presented an aphakic eye, with clear media and a normal fundus. We found in the left eye a characteristic posterior polar cataract. Discission was performed on admission. One month later he was discharged with the eye in excellent condition. Subsequently the patient became unmanageable at home because of a mental derangement, and was sent to the School for Feeble-Minded Children at Spring City, Pa., to be treated for his mental deficiency. So far as we can ascertain, his vision has been maintained in good condition up to the present time.

Developmental deformities of the eye, especially when hereditary in character, offer an interesting field for study and speculation. The literature contains many reports and pedigrees of inherited ocular defects, none of which exhibit their hereditary character more strikingly than do those of the lens. Most observers have been content with an attempt to explain the embryologic changes which may be responsible for the formation of congenital cataracts, and have done but little to explain the hereditary character of these defects.

From an embryologic point of view numerous interesting observations have been made concerning lens defects in the new-born. Von Szily[1] offers three causes for congenital cataract: (1) Inflammation, (2) well-known diseases of metabolism, and (3) essential errors in development of the lens, depending upon so-called "germinal variation." The last cause was supported by observations on a buck rabbit which suffered from partial congenital cataract which he transmitted to most of his offspring. Examinations during the growth of the embryo revealed dismembered scattered groups

of cells at the stage when the lens vesicle was being separated off. These cells developed into fibers, but rapidly degenerated, involving neighboring cell groups in their dissolution. This led to the formation of posterior polar cataract along with several other varieties.

Pagenstecher[2] has shown that there exists a congenital toxic cataract. He produced congenital cataract in 100 per cent. of the offspring of rabbits fed on naphthalin. These were not associated with the separation of the lens vesicle, since the feeding was not begun until the twenty-first day. The result was the formation of many kinds of congenital cataract, not differing in any particular from either the isolated or hereditary cases so often seen. The observation was carried further, however, to the third generation, when the affected rabbits produced normal offspring, showing that the lenticular lesion was produced by a toxin and not by an essential abnormality in the germ-cell of an organic nature.

In relation to the explanation of the formation of posterior polar cataract from an embryologic point of view, MacNamara[3] says, "it was well known that in fetal life branches of the hyaloid artery covered the posterior surface of the lens, and advanced forward over its margin, helping to form the membrana capsulopapillaris. If, from a fault in the development of the eye, these vascular layers persisted after birth, it was apt to give rise to a film of connective tissue extending to a greater or less extent over the posterior surface of the lens, and so forming a zonular cataract. In some instances a small patch alone was left to mark the spot at which the hyaloid artery had passed on to the lens."

Collins[4] holds that the lens-capsule is the product of the epithelial cells lining it. "The hyaline capsule of the lens was at one time regarded as a structure of mesoblastic origin, and the product of the fibrovascular sheath which encircles the lens in fetal life. There is, however, considerable evidence to show that it is really epiblastic in origin, and formed

as a kind of secretion from the epithelial cells lining it." He[5] further states that "the time during which the posterior capsule of the lens has to complete its development is exceedingly short, as the cells forming the posterior layer of the lens vesicle soon lengthen out into lens-fibers and become separated from the capsule. Congenital gaps in the posterior capsule are met with which are bridged over by fibrous tissue formation in the anterior part of the vitreous. This fibrous tissue formation in the vitreous, which sometimes contains blood-vessels from the central hyaloid artery, may extend through the gap in the posterior capsule, an admixture of mesoblastic and epiblastic tissue within the lens capsule resulting."

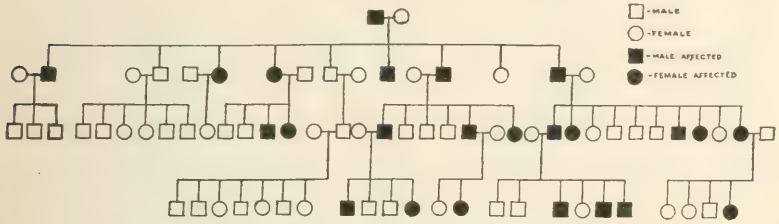
It will thus be seen that observers of congenital cataracts are divided into two schools in their efforts to explain the mechanism by which congenital lenticular deformities are produced: (1) Those who believe the basic cause is toxic, and (2) those who believe there is an arrest of or an alteration in the development of the lens, due to some inherent abnormality in the germ-cell. There is strong evidence to support both theories, and it is probable that neither one singly explains every case of congenital cataract, while in some cases both may be active factors.

Collins (*loc. cit.*) very truly remarks that, "to attribute a condition to an arrest of development offers only a partial explanation of its etiology, leaving the cause of the arrest still to be accounted for." Variations in development can be traced with a fair degree of accuracy to a given group of cells in the embryo, but any inquiry into the cause of this variation leads to a very complex situation about which only surmise exists.

Nash[6] thinks that, "as regards certain developmental defects, there seems to be no reasonable doubt that, on the whole, their descent follows the system of the 'Mendelian dominant,' and that they are due to the presence of special

factors, individuals not possessing these factors being unable to transmit them. The whole body may be influenced by abnormalities in the functions of glandular organs, and through what Garrod has termed unborn errors of metabolism, which latter, at any rate, appear to run in families."

Dr. Charles B. Davenport,[7] after carefully reviewing the pedigree above reported, writes: "The cataract in this family behaves according to expectation as found in other families, namely, as a dominant. We attribute it to mean that there is present in the organism a something which causes cataract. There is a determiner in the germ-cells for that something that causes cataract. The person who has received the determiner for cataract from one parent only will have it in



Pedigree of congenital posterior polar cataract.

half of his germ cells, so that half of his children will show congenital cataract. This expectation is roughly realized in the chart. If in some way or other the determiner for early cataract could be got out of the germ plasm, either by chemical means or others, then those germ-cells should produce individuals without cataract for an indefinite number of generations. But, for the present, so long as these people with early cataract marry, they are bound to perpetuate their trait in half their descendants. Normals need have no fear of marrying or having children."

Loeb[8] divides heredity into three classes: (1) Direct heredity, in which the same disease is found in parent and child. (2) Indirect heredity, in which one or more children

suffer from a disease from which the parents are free, but which is present in one or more ancestors, uncle or aunt. (3) Collateral heredity, in which two or more children suffer from a disease which is not found in either of the parents, or seen in their relatives, with the possible exception of cousins of the same generation.

The scientific study of the causes underlying the recorded facts of heredity has just begun, and so far no authoritative statement has been forthcoming. "Observations and experiments on developed animals and plants," says Conklin,[9] "have furnished us with a knowledge of the finished products of inheritance, but the actual causes and stages of inheritance, the real mechanism of heredity, are to be found only in a study of the germ-cells and of their development. Although many phenomena of inheritance have been discovered, in the absence of any definite knowledge of the mechanism of heredity a scientific explanation of these phenomena must wait upon the knowledge of their cause. In the absence of such knowledge it has been necessary to formulate theories of heredity to account for the facts, but these theories are only a temporary scaffolding to bridge the gaps in our knowledge, and if we knew all that could be known about the germ-cells and their development, we should have little need of theories."

The cause of any pathologic lesion is of interest primarily because it leads more directly to the discovery of methods of cure, and the writers deem it worth while to attempt to determine the etiology of a disease which claims such a large percentage of victims as does hereditary congenital cataract. Are we in such cases dealing with a tendency to germ variation which has exceeded the normal, but which does not assume gross pathological proportions until by chance two people marry, both possessing a tendency to abnormal variation, the cumulative action of their union producing a visible congenital defect? Or are we concerned with a condition

secondary to an intoxication which is itself the result of an abnormality in the internal secretions, or a perversion in the function of elimination? Until such fundamental questions are fully answered we can only deal to the best of our ability with the effect, and contribute our share toward the determination of the underlying cause.

REFERENCES.

1. Von Szily: Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1910.
2. Pagenstecher: Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1911, p. 44.
3. MacNamara: *British Med. Jour.*, Sept. 12, 1891, p. 589.
4. Collins: Developmental Deformities of the Crystalline Lens, *Trans. Sec. on Ophthal., Amer. Med. Assoc.*, 1908, p. 452.
5. Collins: *Ibid.*, p. 426.
6. Nash: Evolution and Disease, p. 47.
7. Davenport, C. B.: Personal communication to the authors.
8. Loeb: Hereditary Blindness and Prevention, *Annals of Ophthal.*, January, 1909, vol. xviii, No. 1, p. 8.
9. Conklin: Heredity and Environment, p. 104.

ADDITIONAL BIBLIOGRAPHY.

Baker: Infantile Cataract, *Jour. Amer. Med. Assoc.*, September, 1892, p. 278.
Bastard: *Thèse de Montpellier*, 1850.
Baudon: *Recueil d'Ophtal.*, 1878, p. 9.
Chance: *Archives of Ophthal.*, 1907, vol. xxxvi, p. 505.
Derby: *Trans. Amer. Ophthal. Soc.*, 1890, p. 722.
Fromaget: *Gaz. hebd. d. Sc. Méd. de Bordeaux*, July 30, 1893, p. 368.
Gunn: *Ophthal. Review*, 1898, vol. xvii, p. 129.
Hansell: *Ophthal. Record*, June, 1896, p. 488.
Nettleship: Royal London Ophthal. Hosp. Reports, vol. xvi, iii, p. 179; *ibid.*, vol. xvi, iv, p. 389.
Thomson: *Lancet*, 1889, ii, p. 1282.
Wood: *Ophthal. Record*, April, 1906.

DISCUSSION.

Dr. S. Lewis Ziegler, Philadelphia: Some of the questions involved in the problem of heredity are difficult of solution, but I think that it is possible to favor, to a certain extent, the hypothesis of an underlying organic lesion that exerts some specific influence on either the internal secretions or the lymph secretion, thus creating a perversion of the chemistry of these secretions. Pagenstecher's experimental feeding of naphthalin to rabbits after the twenty-first day of embryonic development, and the production of

cataract thereby in every case, tends to confirm the toxic origin of lenticular disturbances in the newborn. His further study of these animals in subsequent generations showed that this cataract was only a temporary aberration, due to the ingestion of a toxic chemical substance, and that there was no underlying organic lesion present. These studies, therefore, by negative provings, furnish very convincing evidence favoring the hereditary side of the question.

In regard to the postoperative inflammation in one of the cases recorded in the paper, this seemed to be of endogenous origin, as the wound was closed. It occurred some days after the operation, and was not an immediate effect of that procedure. Whether this was metastatic or not, we were unable, of course, to determine. The boy, as stated in the paper, had innocently acquired gonorrhea at school, and had suffered severely from its systemic invasion.

FAMILY DEGENERATION OF THE MACULA LUTEA, WITH A SUGGESTION AS TO ITS CAUSE.

BROWN PUSEY, M.D.,
Chicago, Ill.

Medical literature contains the records of observation of eight families in which two or more members of the same family have been afflicted with lesions of the macular region of the retina, with onset at about the thirteenth year, without known cause or suggested pathology, and without general lesions.

The first report of such a family affliction that I have found in a fairly careful look through the literature is that of Batten.* In a short clinical note he reported two brothers with the affliction coming on in the thirteenth year. In 1909

* Batten, R. D.: *Trans. Ophth. Soc. U. K.*, 1897, xvii, 48.

Stargardt* reported in detail two such families. One family consisting of four persons, all of whom were afflicted; the other family consisting of five persons, three of whom were afflicted. In the same year Jennings† reported such a family with three members afflicted. Lutz‡ has reported such a family, six members of a family of nine being afflicted. In 1913 Stargardt§ reported another family, three members of which were afflicted. In 1914 Darier|| reported two such families.

A family with such macular lesions, with onset at the twelfth or thirteenth year, came under my observation about two years ago, and I make this report because of the general interest of the condition, and because I believe an associated symptom in the patients of this family suggests a clue to the cause of the affliction.

A FAMILY OF EIGHT CHILDREN.

(1) Mrs. H., aged thirty-three years. Her eyes were examined when she was ten years old by a competent oculist who pronounced them normal. In the fall of her thirteenth year, when she returned to school, she found that she could not see writing on the blackboard from the first seat; she could not read the notes of her music; she could not read unknown words of ordinary book print; she could read known words of fair-sized print. In the spring of that year there was no disturbance of vision. From the fall of the thirteenth year the vision remained unchanged until lately, when the central vision became noticeably worse.

At present the vision is $\frac{6}{60}$, each eye, with correction, −4.50 D. sph. The visual fields for form are normal; for colors the fields are normal, except at the center, where there is a relative negative scotoma for all colors except red, for which the scotoma is absolute. She says that distant ob-

* Stargardt: *Graefe's Arch.*, 1909, lxxi, 534.
† Jennings, J. E.: *Amer. Jour. Ophth.*, 1909, xxvi, 296.
‡ Lutz, A.: *Klin. Monatsbl. f. Augenheilk.*, 1911, xlix, 699.
§ Stargardt: *Zeitschr. f. Augenheilk.*, 1913, xxx, 95.
|| Darier, A.: *Clin. Ophth.*, 1914, xx, 3.

jects are seen better with a dim light than with a bright one—at dusk she can see a human figure a block away; in a bright light she cannot. The eyes externally are normal. The pupils react promptly to light when light is thrown on the periphery of the retina; they react very sluggishly when light is thrown on the macular region, the contraction of the pupils being so slight that ophthalmoscopic examination of the macular region is not at all interfered with. Intraocularly the eyes are normal, except at the posterior poles. Ophthalmoscopically the macular region presents the picture commonly described as "moth-eaten macula." In this region, in a horizontally oval area of about two disc diameters, there are small pigment splotches in a dirty brownish background. There is no swelling or depression of the tissue, and the choroid appears to be uninvolved. The picture is that most commonly seen in elderly arteriosclerotic people, and described as senile degeneration of the macula. The optic papillæ show slight temporal whitening.

(2) A brother, aged thirty-one years: It was not possible to examine this man. There is no disturbance of vision.

(3) A sister, aged twenty-nine years: In this sister the visual disturbance came on in the thirteenth year. Her symptoms are essentially those of the senior sister, and the ophthalmoscopic findings are the same. She is slightly myopic.

(4) A sister died at ten months.

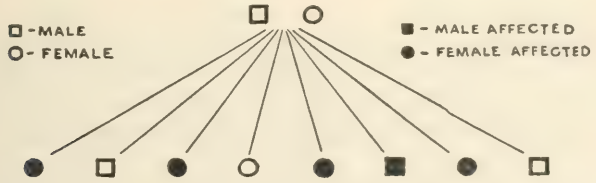
(5) A sister aged twenty-five years: The visual disturbance began in her thirteenth year. The symptoms and ophthalmoscopic findings in this woman are those of the senior sister, with the exception that the optic papillæ are normal. She is not myopic.

(6) A brother, aged twenty-two years: The visual disturbance came on in the twelfth year. In this brother the symptoms and ophthalmoscopic findings are those of the senior sister, with the exception that the optic papillæ are normal. He is not myopic.

(7) A sister, aged nineteen years: The visual disturbance came on in the thirteenth year. In this sister the symptoms and ophthalmoscopic findings are those of the senior sister, with the exception that the optic papillæ are normal. She is not myopic.

(8) A brother, aged fifteen years: Not affected.

The above family history may be summarized as follows:



In the investigation for a possible cause it was immediately apparent that the family was one of more than usual intelligence, which removed it from that group of observed cases of macular degeneration associated with degeneration of the central nervous system. There was no history of a similar affliction in their antecedents. There was no consanguinity.

In questioning the senior sister it developed that she and other members of her family were subject to cardiac attacks. She and other members of the family were referred to Dr. Charles A. Elliott for general examination, and I am indebted to him for the following notes:

Mrs. H., aged thirty-three years. Patient seeks relief from—(1) Failing vision; (2) pain in the cardiac region; (3) fainting attacks; (4) headache. She remembers having had attacks of pain in the cardiac region since she was fourteen, but they first became severe at the time of her pregnancy, twelve years ago. The attacks may be slight or very severe. The slight attacks are described as a peculiar sensation in the cardiac region which lasts about five minutes and recurs every three or four days. She is conscious of the pain, but it does not interfere with whatever she may be doing. The severe attacks come on less frequently (about ten attacks in twelve years). They are of sudden onset and cramp-like in character, radiate to the left forearm, and are always accompanied by a fear of impending death. The attack gradually wears away, lasting about twenty minutes, after which she feels exhausted, but is soon able to go about her work as usual. Fainting attacks first appeared when she was thirteen

years old, then at fifteen, and since then about once a month. She is unconscious, but is readily revived. Both the cardiac pains and the fainting attacks occur independently, but the latter are usually accompanied by the former.

Previous illnesses: Scarlet fever at eleven—very sick; typhoid fever at fifteen—eight weeks in bed, convalescence slow, no complications; peritonitis at sixteen—unknown origin, sick two months; pneumonia at seventeen—sick five weeks, no complications. Menstruations painful, otherwise normal. Once pregnant, normal, child now twelve years old, living and well.

Physical Examination.—General: A well-nourished, neurasthenic woman. Face flushed, mucous membranes anemic, tongue coated, many teeth decayed. Skin moist and soft; no edema; no adenopathy. Tonsils enlarged. Cardiovascular: Pulse soft, easily compressible, vessels soft. No evidence of arteriosclerosis. Apex-beat not visible. Dulness 9 cm. to right of median line, upper border at fourth rib. Everywhere two tones, no murmurs. Second tone clear, not accentuated. Blood-pressure: systolic, 113; diastolic, 75. Lungs: Diaphragm stands at normal level. Excursion good. Resonant throughout; no râles. Abdomen: Pendulous, soft, relaxed. Caput coli distended and tender. Descending colon contracted and tender. Liver: five fingers in breadth, not palpable. Spleen not palpable, area of dulness present, normal. Kidneys not palpable. Reflexes everywhere present and normal. Urine: single specimen, 1010, acid, trace of albumin present, no sugar, acetone, casts, or blood present. Calcium oxalate crystals present. Blood: Erythrocytes, 2,944,000; leukocytes, 14,000; hemoglobin, 87 per cent. (Dare). Pulse, 96. Temperature, 99.8° F. Wassermann negative.

Family History.—Father, aged sixty-four years, living, has had some cardiac distress upon exertion. Eyes normal; no headaches or fainting spells.

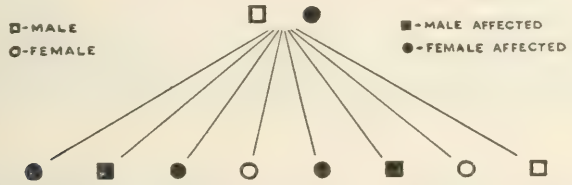
Mother, aged fifty-three years, living, has had sharp, shooting pains in the cardiac region since twenty, which she calls "neuralgia," and which recur in attacks at about one month's interval. Eyes normal; has headaches and suffers from tic douloureux. Does not faint.

Brothers: (1) Aged thirty-one years, had cardiac pains

while in college, and fainted about five times when young. No headaches. (2) Aged twenty-two years, has occasional attacks of pain in the cardiac region, which are not so severe as those of patient. Has headaches. Does not faint. (3) Aged fifteen years, an epileptic for several years. No eye trouble. Does not have headaches, nor does he have cardiac pains. Wassermann negative.

Sisters: (1) Aged twenty-nine years, an actress who has attacks of sharp cardiac pains and headaches and has occasional fainting spells. (2) Died in infancy. (3) Aged twenty-five years, has severe attacks of cardiac pain which incapacitate her for work, and for which she goes to bed for relief. (4) Aged nineteen years, has headaches. No heart attacks. Does not faint.

The history of cardiac pains in this family may be shown as follows:



We have then here a family which shows a marked tendency to cardiac angina, beginning in early life. Of seven brothers and sisters who lived to adult life, five of them were affected. The mother of these children also gives a definite history of similar trouble, and the father a less definite one. Such attacks of cardiac distress are commonly caused by arteriosclerosis, and there can be little doubt that this family shows a marked tendency to the premature occurrence of this condition.

Of these seven brothers and sisters, five show also degeneration of the macula lutea. The ophthalmoscopic picture in this group of cases is that of senile macula, a condition recognized as due to arteriosclerosis. Putting these two facts together, there would seem to be little room for doubt that the macular degeneration in this family is a manifesta-

tion of the same pathologic process that is responsible for the cardiac disturbance; that is, a premature arteriosclerosis to which the family shows a marked common tendency. The probable etiology of the condition in this family suggests that premature arteriosclerosis is the cause of the condition in general.

DISCUSSION.

DR. JOHN E. WEEKS, New York: I should like to ask Dr. Pusey where he places the defect in the vascular system. It would seem to me that this is a picture of the degeneration that takes place when the capillaries of the choroid particularly are involved.

DR. W. H. WILDER, Chicago, Ill.: I should like to know whether Dr. Pusey found any evidence of sclerosis of the retinal arteries.

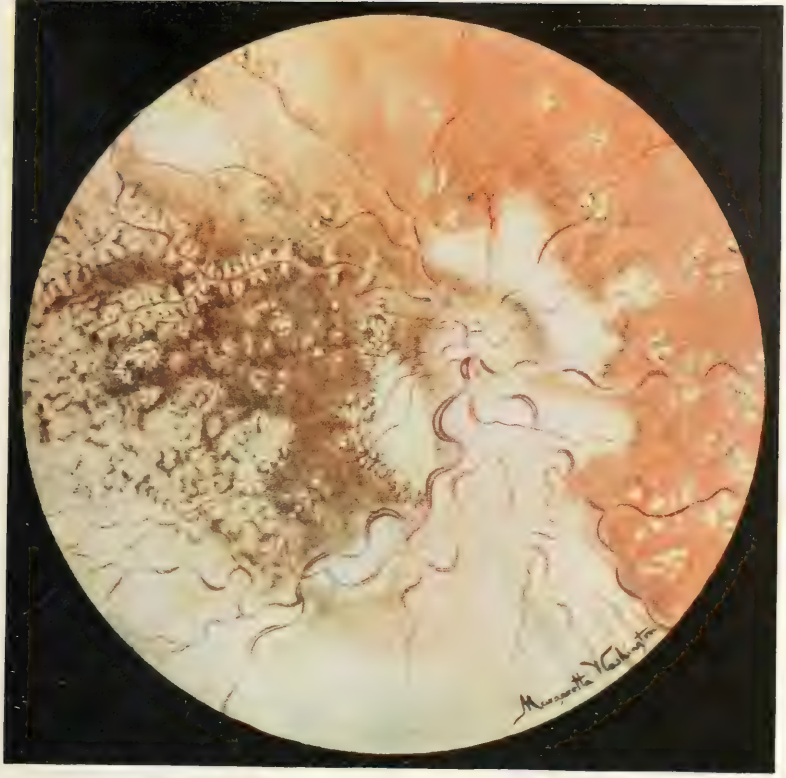
DR. BROWN PUSEY, Chicago, Ill.: I would say that the pathology in these cases is arteriosclerosis of the vessels—terminal branches—of the macular region. There was no disturbance in the choroid. The eye pathology is limited to the macular region of the retina, with the exception of temporal atrophy in the nerves in the oldest patient.

RETINAL DISEASE WITH MASSIVE EXUDATION AND AN ALLIED CONDITION: REPORT OF TWO CASES.

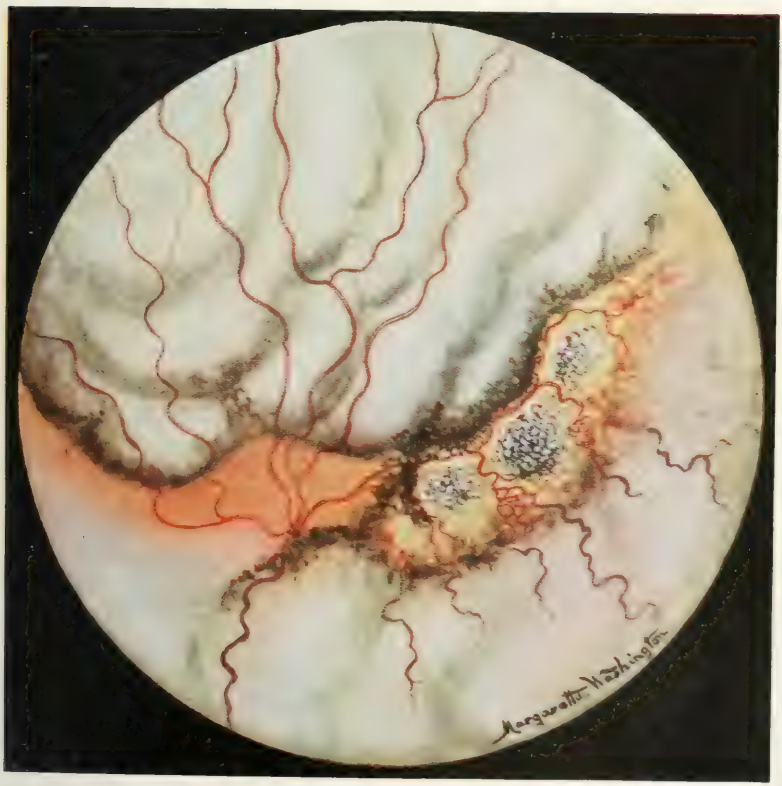
WILLIAM ZENTMAYER, M.D.,
Philadelphia, Pa.

The notes of the first case are solely clinical, but the ophthalmoscopic picture and the anamnesis are so typical as to leave no doubt but that it belongs to the first group of Coats' classification of Retinal Disease with Massive Exudation, namely, those without gross vascular changes.

M. T., Italian boy, nine years of age, was brought to Wills Hospital February 15, 1915, because of failing sight, of which



Case 1.—Right eye.



Case 2.—Left eye.
Retinal disease with massive exudation.

he had complained for four months. Up until this time he went to school regularly and could read and write. He still sees fairly well in daytime, but cannot go about at night. At two years of age he had a mild attack of measles, and between the age of six and seven years he had an indefinite illness lasting two and one-half weeks. There is no history of trauma to the eyes, nor has there been any inflammation or pain in the eyes. There has been no ear disease. The lad is well developed physically, but is mentally precocious and inquisitive. He is the fifth and only living child of a family of six. The first child died at the age of two and a half years; the second lived eight days; the third was born dead; no data as to the fourth; the fifth is the patient, and the sixth died when three and a half months old. The mother died at the birth of this child. The father is living and well and denies venereal infection.

The physical examination of the child is negative, as are also the blood, Wassermann, and the tuberculin tests. The urine has a specific gravity of 1018, and contains neither albumin nor sugar.

V. R. E. = $\frac{3}{60}$; L. E. = l. p. The right pupil measures 4 mm. and is sluggish to light. The cornea is clear, the anterior chamber of normal depth. T., 18 mm. The left pupil measures 5 mm., and is immobile to light. The pupillary pigment border is very broad, and the iris stroma is somewhat thinned. T., 22 mm.

The ophthalmoscopic examination of the right eye shows the vitreous filled with dust-like opacities. The papilla is prominent and overcapillary. There is an opaque, dense, greenish-white, prominent mass extending from the region of the ora serrata up to the lower border of the papilla, and over the temporal portion of the fundus as far forward as can be viewed with the ophthalmoscope. The surface of the inferior mass is undulating, as evidenced by the course of overlying vessels. The margins of the mass do not end abruptly, but merge into the surrounding retina. In places the edge is skirted by small, irregularly round dots, which coalesce to form a broad fringe. Groups of similar dots are found elsewhere in the fundus, and are particularly marked along the course of the superior temporal artery and its branches. These latter areas are not unlike the patches seen in retinitis

circinata. There is a second opaque area surrounding the upper-inner margin of the papilla. The vessels supplying the affected areas, especially the veins, are all enlarged and tortuous. In places they are slightly veiled, but are not actually covered by the exudate. The surface of the mass is quite prominent. No hemorrhages are visible.

In the left eye the retina is detached in lobulated areas extending forward from the papilla to the ora serrata. In the lower part of the fundus, between two of the lobes, the retina is still in position, and a choroidal reflex can be obtained. The surface of the retina is gray, and is in places flaky and glistening. Over the inferior portion, where the detachment is less prominent, there are hemorrhagic extravasations. The detached portions of the retina are thickened and "set" in appearance, and the separation is apparently not due to serum.

The boy was under observation for about six weeks, and during that time little change occurred in the appearance of the fundus, but vision continued to fail despite the energetic use of mercury. About three months after his discharge from the hospital a telegram was received from his father stating that the boy had become blind.

The one unusual feature in this case is its bilaterality. In the 23 or 24 cases collected by Coats falling within this group, both eyes were involved in but two instances. And in all the typical cases I have since found in literature the affection was, at the time of the reporting, unilateral. A point of interest is the differential diagnosis from neuroepithelioma. In this particular case the characteristic changes of massive exudation in the right eye made the diagnosis clear. But where the condition is confined to one eye and appears in the form, or at the stage, seen in the left eye, the diagnosis is difficult. The age of the patient, the glistening areas on the surface of the retina (as cholesteatomata are usual in massive exudates), and possibly a greater tendency to hemorrhagic extravasations may aid in the diagnosis.

The second case was a girl, aged eight years. Two days

before she was brought for examination a school-mate had discovered the peculiar appearance of the child's right eye. Six weeks before she had had a mild attack of measles. Since infancy there has been otitis media purulenta on the left side, and recently also on the right. She was a sturdy child. Physical examination was negative. Neither the tuberculin nor the syphilitic tests were made, as at the time the child was seen these methods of diagnosis were not in general use. The father and mother are living and healthy. There are two other children, aged, respectively, five years and one year.

In the right eye the pupil is semidilated, and from behind the transparent lens there is a suspicious reflex. Focal illumination shows this to be due to a totally detached retina, which has been forced forward to a position just behind the lens. The detachment is in the form of four lobes separated by deep sulci. The retina is gray, thin, and mottled. Some of the normal vessels can be made out, but no new-formed vessels are visible. On sudden movements of the globe a slight tremor is at times observed in the retinal mass. Tension is a shade elevated. Transillumination gives a good reflex.

The patient was kept under observation for six months, when, owing to symptoms of secondary glaucoma, the eyeball was enucleated. In operating, the globe was perforated, giving exit to a serous fluid containing cholesterin. On sectioning the globe the subretinal space was found occupied by a similar fluid. The surface of the choroid presented small, nodular elevations.

Pathologic Findings.—The microscopic study was made by Dr. Harold Goldberg and also by Dr. Allen J. Smith. The cornea is normal, except for a slight proliferation of the endothelium. The anterior chamber contains blood. The iris and ciliary body are somewhat atrophied. Hemorrhagic extravasations are scattered throughout the ciliary body. The choroid is thickened, and its vessels show hyaline thickening. There is a universal pathologic detachment of the retina. Many small hemorrhages are present in the inner nuclear and in both plexiform layers of the retina. There is some turgescence of the vessels. The fiber layer is slightly thickened, as is likewise the ganglion-cell layer from glial

proliferation and edema. The optic nerve shows a moderate grade of neuritis, with a slight excess of young connective-tissue elements.

There may be some question as to the propriety of including the second case in the category of "allied conditions" to retinal disease with massive exudation, but it seems justifiable, inasmuch as the essential feature in both is extensive subretinal and intraretinal hemorrhages, and as clinically there was a striking similarity between this eye and the left eye in the first case, in which the fellow-eye showed unmistakably massive exudation in the retina. The microscopic changes, while not identical with those met with in the latter condition, probably represent a different stage of the disease. The absence of changes usually met with may be explained by the fact that the hemorrhages were too recent for degeneration and organization to have taken place. The thickening of the fiber-layer and of the ganglion-cell layer from glial proliferation and edema indicates a step in the direction of the changes met with in this disease.

As Coats remarks, little is known of the early stages of the disease, as most of the cases which have been examined have been of very long standing. In his argument for the supposition that the condition is due to slow organization of hemorrhages, Coats brings forward as clinical evidence the conclusions of Nettleship, stated by that clinician in discussing a case of Snell's almost identical with my second case, that "there is a class of cases where, in children, gross detachment of the retina occurs in connection with thick plates, sometimes white, sometimes yellow, in or behind it, with an appearance as indicating subretinal hemorrhage." In one such case that he had examined there was a sediment in the subretinal space, consisting chiefly of fat-globules. As to the causation, nothing was known, but he suggests that there might be some connection between the detachment and an exanthema. In a "Report of Two Remarkable Cases of

Choroidoretinal Disease in Children Without Assignable Cause," Nettleship gives the notes of a case which to-day would be assigned to the first group of massive retinitis, and says that the hypothesis that the condition resulted from an extensive intraocular hemorrhage during whooping-cough received support, especially from the fact that some blood-patches were actually present in the eye. It is interesting to note further that Collins and Mayou consider retinitis circinata and massive retinitis to be allied affections.

DISCUSSION.

DR. G. O. RING, Philadelphia: I should like to place on record, in association with this case, one now in the wards of the Episcopal Hospital, Philadelphia. It is in an attractive girl of twenty-two, who has an absolutely normal right eye and an entirely negative personal and family history, barring a slight enlargement of one of the cervical glands. She is negative to all types of examination, looks perfectly well, and has, to external apperance, a normal left eye, but with a very extensive exudation, beginning on the nasal side, and involving the major portion of the retina. There are three large masses of this exudate, from which are suspended thread-like filaments. The case has resisted treatment, and has remained for a year in practically the same condition.

DR. W. F. MITTENDORF, New York: I should like to speak of a young lady, the picture of the fundus of whose eye reminds me of the illustration Dr. Zentmayer has shown. This was also evidently caused by hemorrhages, which began to cicatrize, and eventually the whole retina became almost one mass of organized tissue. The lens finally became opaque. Otherwise the girl enjoys good health. All tuberculin and other tests have been negative, and I suppose it is one of the few cases of profuse hemorrhage that we sometimes find in young girls at the age of puberty.

SPONTANEOUS REATTACHMENT OF THE RETINA AFTER TWENTY-TWO YEARS' DURATION; REPORT OF CASE WITH REMARKS.

A. E. DAVIS, M.D.,
New York City.

A considerable number of isolated cases of spontaneous reattachment of the retina have been reported, but in none can I find where reattachment has taken place after having remained detached for so long a time (twenty-two years) as the case here reported.

The nearest approach to it in point of time is a case by Franke,* of eleven years' duration, which did not yield to treatment, but spontaneous cure took place at the end of this period.

F. B., aged eighteen years, first seen August 12, 1897, with history of a fall from a carriage when six years of age and striking the left side of his head. He did not notice that the sight was affected at the time, and it was not for some weeks afterward that the vision became bad in the left eye; he being able to see only indistinctly downward and to the left. Patient has always been in delicate health, and is tall, thin, and pale. History of tuberculosis in the family. No history of syphilis or rheumatism.

V. R. E., sph. + 1 D. = $\frac{20}{15}$; L. E. = fingers at two feet down and out. Field limited, corresponding to the detachment. Ophthalmoscope shows normal fundus right eye, H. + 1.50 D.; left eye, detachment of the retina involving almost the entire lower half, extending from the nasal side along the horizontal meridian to the disc, and then from the optic disc toward the temple and slightly upward. The

* Franke: *Klin. Monatsbl.*, cited by E. Thomson, *Ophthal.*, vol. viii, p. 105.

detached portion is flattest nearest the disc, and highest at the lower part of the fundus in the vertical meridian, where the retinal vessels can be seen plainly with + 9 D.; down and nasally with + 6 D.; down and temporally with + 4 D. A narrow white band extends horizontally across the fundus, slightly inclined from down and in, up and out, and demarcates the detached from the attached portion of the retina. The tension is slightly plus.

The present case is of special interest, it seems to me, because of two or three unusual features. The first and most important one is the reattachment of a retina after it has remained detached for twenty-two years. Second, that the reattachment was spontaneous, without any local treatment. Evidently the detachment in this case occurred when the patient was between six and seven years of age, when he noticed the very poor vision following an injury. This was eleven years before he came under my observation; and he remained under my immediate observation for eleven years until sent to the country, the detachment remaining about the same while under observation. This made a period of twenty-two years of detachment. The patient remained in the country for six years without the eyes being examined, improved much in health, and gained many pounds in flesh.

On reëxamining the eye after his six years in the country the retina, to my great surprise, was found to be completely reattached, though the field was not restored. But after a detachment of such long duration a restoration of the field was not to be expected. The field, in fact, was almost identical with that taken when the retina was detached. The narrow subretinal band of demarcation, which had made the boundary between the attached and detached portion of the retina, had become a little broader, with a few pigment specks at the lower border of the band, while the band on the nasal side had spread out at the periphery into a wide patch of choroidal atrophy. Several other narrow white bands

were to be seen beneath the reattached retina, especially on the temporal side and below, while down and in from the optic disc were two or three small, round, clear-cut patches of choroidal atrophy. In addition, just below and to the temporal border of the optic disc, and encroaching upon it, was a slightly elevated, yellowish-white exudate resembling in a marked degree a tubercle. The ophthalmoscope showed some dust-like opacities in the vitreous, and a hyperopic refraction of 1 D. in all parts of the fundus, there being no difference of level over the reattached retina from that of the other portions; and it is interesting to note at this point that the patient, before going to the country, had developed tuberculosis, the apex of the left lung being involved.

The tuberculin tests, both the von Pirquet and the subcutaneous injections of old tuberculin, gave positive reactions, and the therapeutic injections of bacillen emulsion mixture gave reactions of a local and general character, but no focal reaction. Because of a tendency to cough and some irritation at the apices of the lungs, the bacillen emulsion treatment was discontinued, the country air, good food, and regular hours being depended on for cure of the consumption, and we trust a permanent cure of the detachment, which has remained reattached now for seven months. A Wassermann test proved negative.

It is interesting to speculate how a spontaneous cure is brought about in these patients. In the present case I am of the opinion that a choroiditis of a tubercular nature developed subsequently to the traumatic detachment, and that, with the improvement of the patient's health when in the country, the choroiditis subsided, and in the healing process the retina was bound down to the choroid. The old patches of choroiditis and the white streaks under the retina all point to this method of cure. Casper, Leber, Praun, Imre, and others consider the white subretinal streaks as the final stage in the cure of traumatic detachment. In my own case not

only were these subretinal streaks present, but old patches of healed choroiditis, both factors, in my opinion, entering into the cure.

A case reported by Fano (1876), and cited by Ernest Thomson,* though of not so long duration, has several points of similarity to the one here reported: "A soldier, after a blow on the orbit, developed an inferior detachment. Five years later he returned to Fano, who found the detachment had entirely disappeared, leaving complete choroidal atrophy at its site. Iritic adhesions were also found at this time, and the author draws the conclusion that a severe iridochoroiditis had resulted in a destruction of the choroid and a soldering down of the retina to the choroid."

The general consensus of opinion respecting these cases is in accord with the statement of Weeks: † "Spontaneous recovery takes place only in a very small percentage of cases. This is most apt to occur in detachment following traumatism not associated with high myopia, detachment with albuminuria, detachment due to inflammatory choroidal exudate, to disturbed circulation, and to diseases of the orbit, rarely in detachment accompanying myopia."

Ernest Thomson, ‡ in a general review on Detachment of the Retina, gives a most excellent résumé of the whole subject, as to pathology, diagnosis, treatment, and prognosis, and a review of the literature up to that time, with comments on the interesting cases.

Under the heading of spontaneous cures he states: "I am sufficiently skeptical, and at the same time forced by necessity, to include cases where there may have been treatment of a simple nature, such as rest in bed, bandaging, atropin, and so on, but no active medical or surgical therapeusis. The study of spontaneous cures is of as much

* Thomson, Ernest: *Ophthalmoscope,* London, 1910, vol. viii.
† Weeks: Text-book of Ophthalmology, p. 486.
‡ Thomson: *Ophthalmoscope,* 1909–1910.

moment as that of cures following treatment, because the latter is so notoriously ineffective, and because there can be no doubt that many, many cases reported as cures, following more or less complicated treatment, would have become cured if left to themselves or treated by rest alone."

Thomson speaks of spontaneous cure as meaning spontaneous reattachment, with or without recovery of function. He remarks as to the contradictory statements of the early writers on the subject of spontaneous or of any cure of detachment of the retina, which holds even to the present-day writers, some reporting many cases, some none at all. He cites Hirschberg, who in 1870 had not seen a single spontaneous cure out of 113 detachments. This author later reported four or five cases of spontaneous cures.

"Abadia, in 1884, denied the permanence of reattachment, and in 1900 stated, in thirty years' practice, he had never seen a single spontaneous cure. Lagrange, in 1899, stated he had not seen one single myopic detachment cured. Galezowski, in 1887, only admitted one spontaneous cure out of 784 cases of detachment."

In spite of the above pessimistic reports, Thomson was able to cite the reports on many cases of spontaneous cure even in myopic patients, for instance: Steffan, 1 case; Galezowski, 3 cases; Armaignac, 1 case; Hirschberg, 4 cases; Donburg, 3 cases; Kopff, 1 case; Chevallereau, 1 case; Sulzer, 1 case; Higgins, 1 case; Horstmann, 5 cases. These cases of Horstmann were carefully reported and kept under observation for years, and there seems to be no doubt as to the permanency of cure.

As to the spontaneous cure of detachment in nephritis of pregnancy, Thomson states: "It is impossible to avoid a feeling of certainty that cases of detachment occurring in albuminuria of pregnancy represent a morbid process differing in some essential way from that which exists in myopic and even traumatic non-myopic detachments." Traumatic

detachments are supposed to be the most favorable cases for spontaneous cure, and a number of such cases are cited by Thomson in his review. It is interesting to note here also that Muglich,* in 1891, collected 136 cases of spontaneous cure of detachment from all causes.

Prognosis.—Although the prognosis is considered more favorable in traumatic cases by the vast majority of surgeons, Thomson, after his exhaustive review of the subject, states he is not convinced that the evidence is conclusive of a better prognosis in traumatic than in myopic cases. And he states further that the statistics of spontaneous cure are of the most contradictory character, with which statement one must fully concur on reading the different reports of the cases cited in literature. Also, one is inclined to agree with him in another observation: that the treatment of detachment is to a certain extent a game of chance.

In his general conclusions Thomson, in making a comparison of cures obtained by operative methods, including subconjunctival injections, with the non-operative methods, found 10 per cent. of cures were secured by operative methods and 7.2 by the pacific or non-operative method. With this favorable showing by non-surgical methods, he is of the opinion that we ought to leave the decision as to the method of treatment to the patient, without too much pressing forward of an operation. Unless trephining the sclera, the newest operation for detachment, should prove more decidedly successful in bringing about cures than previous operations devised for this affection, I am inclined to favor the non-surgical treatment. However, the patient should, in a measure, after the two methods have been placed before him for consideration, be allowed to choose which method of treatment should be followed.

* Muglich: Inaugural Dissertation, Marburg, 1891.

DISCUSSION.

Dr. T. Y. Sutphen, Newark, N. J.: It is my personal conviction that reattachment of the retina is dependent upon an existing adhesive condition of either the choroid or the retina. Whether this occurs as the finale to the original causation of the detachment or results from operative interference or the presence of chemical sources of irritation it matters not; the membranes must adhere and must remain in apposition for a certain length of time, to become permanently adhesive. In this way, it seems to me, can be explained the cases of spontaneous reattachment, in the earlier stages, as well as those resulting from operative interference. I cannot help believing that there is a stage of more or less adhesive tendency in all cases of detachment of the retina, and this would explain what we see in detachment of the upper portion of the retina changing into detachment of the lower portion, with subsequent reattachment of the membrane in its upper portion, and in some cases a disappearance, also, in the lower portion. In a case of complete detachment that I operated upon by scleral puncture many years ago, in which good vision was retained for twenty years, until the man died, as well as in the case of deWecker's, upon which he operated a year or two earlier, and which was seen by members of this Society, I have always felt that either the psychologic moment was struck for an adhesive inflammation, or this condition was caused by the operative interference; and so I would naturally judge that Dr. Davis's case found the psychologic moment of stickiness, either through some tubercular process or otherwise.

Dr. Arthur J. Bedell, Albany, N. Y.: I should like to cite the case of a woman, aged thirty-seven, who was first seen September 6, 1909. The vision of the right eye was $\frac{6}{3\,0\,0}$, and of the left, $\frac{2\,0}{3\,0}$ + . She had a pearly gray cataract in the right eye, the media of the left being perfectly clear. One year later the vision of the left eye had decidedly dropped, so that the right cataract was removed with a broad corneal section, a conjunctival flap, and a small iridectomy. Two weeks later a V-shaped capsulotomy was performed, and thirty days after operation vision, with sph. + 13 D., equaled $\frac{2\,0}{1\,5}$.

On September 24, 1912, the left lens was removed in the same way, and with sph. + 12 D. vision was $\frac{20}{15}$ −. Everything progressed nicely until August 7, 1914, when the patient came complaining that for the past three weeks the vision of the left eye had been failing. On examination it was found that she could see fingers in the lower outer field only. There was a definite and distinct detachment of the lower half of the retina. Later, she had only light perception, and, on the twenty-eighth of the month, saw Dr. de Schweinitz, who suggested the usual treatment.

She was placed under general observation, which included a rather restricted diet, because of an increased arterial tension. Her general health improved in every way, and on January 5th of this year the vision of the left eye had returned to $\frac{20}{50}$, and it was impossible to find the detachment. When last seen, June 14th, the media of the eye was clear; and, with a correcting lens, vision equaled $\frac{20}{30}$, combined with a sph. + 3 D., = J. 1.

The patient's sister, of about the same age, has had both cataracts removed.

This case is reported because the detachment had been verified by another observer, and because the loss of vision was complete. Without local medication, the retina has returned to its normal position, and full function has been restored to the eye. I make no attempt at explanation, feeling that any one who has gone through a similar experience is competent to draw his own conclusions.

DR. W. GORDON M. BYERS, Montreal, Canada: The following case is of interest because of the long period—over ten years—during which the cure has been observed. I think it may properly be called a spontaneous reattachment, because no local measures were employed, and the rest was more or less imperfectly carried out.

F. C., in every respect a perfectly healthy man, aged thirty-three, consulted me on August 22, 1904, for a blurring of vision in the right eye which had followed immediately upon a blow from a green orange, well back upon the right temple, during an improvised game of hand-ball two weeks previously.

On examination the left eye was found to be entirely

normal, with a visual acuity of $\frac{6}{5}$, with a -0.25 cylinder axis 180. In the right eye there was a large detachment of the retina, which attained its greatest prominence near the center of the superior temporal quadrant of the fundus, and gradually receded downward and outward. In the macular area the retina was also thrown into a few vertical striæ, but these appeared to have no direct connection with the larger separation. There were no vitreous opacities, but the reflex from behind the retina was very dark, and bespoke a more or less bloody extravasation.

About two weeks later the detachment had extended to about the middle line, but from that time on it receded, in spite of the fact that the patient remained in bed for only a short time at the outset, and afterward went about his office work much as usual. By November 15, 1904, the detachment had completely disappeared. During this period the patient's central sight, $\frac{6}{18}$ at the time of his first visit, rose to $\frac{6}{12}$, while frequent notes mention that the tension of the globe was normal, and that he complained of seeing things yellow. His peripheral vision at the outset showed a marked constriction in the nasal half of the field, and an apparent loss of function in a narrow band of about 10 degrees, involving practically the whole of the periphery of the retina.

The man returned in January, 1905, with a relapse of the detachment involving the whole of the temporal half of the retina, although this time it was not so pronounced as previously. The tension was now minus, and the central vision was reduced to $\frac{6}{18}$, without any disturbances of the field of vision. Under rigid rest in the dorsal position the retina rapidly regained its normal situation, and there was no separation to be seen on May 9, 1905. At this time, however, I made the following notes: "Under mydriasis, the right eye shows a milky appearance in the macular area and, to a less extent, over the whole of the temporal side of the fundus. Small whitish dots are seen in the macular area and along the course of the superior temporal artery. In the extreme periphery upward one makes out two small, rounded areas of pigmentation, surrounded by hemorrhagic extravasations, and, downward and outward, a couple of dots of pigment accumulation."

On July 3, 1915, I made a further examination of the patient under mydriasis. The left eye was normal, as previously, with the same error of refraction and degree of vision; but over practically the whole of the outer half of the temporal side of the right fundus one could see patches, for the most part of considerable size, in which the retinal pigment epithelium had accumulated in masses of varying density, and beneath the retina one still perceived a milky appearance, which suggested a deposition of connective tissue. The central vision reached $\frac{6}{5}$, with sph. $-$ 0.75 ⊂ cyl. $-$ 0.50 axis 90°, and the peripheral vision was full, except at two points inward, which corresponded to areas in which the pigment disturbances were most marked.

DR. WALTER E. LAMBERT, New York: I should like to record a case of spontaneous reattachment of the retina which occurred in my practice. A woman, sixty years old, had had both eyes operated on for cataract, with excellent result— $\frac{20}{30}$ vision in each eye. Three years after the operation in the left eye she appeared at my office with the statement that vision had failed in that eye, and there seemed to be something floating before the eye; V. = fingers in lower field. I found a complete retinal detachment in the lower quadrant. I took her to the Infirmary, ordered rest and the usual treatment. I suggested an operation, trephining, but her family were not anxious to have this done, and I did not urge it. After several weeks I gave the case up as hopeless, and the patient returned to her home at Stamford, Conn., with the injunction to be careful. Three months later, May 25, 1914, she appeared at my office with complete reattachment and restoration of the function of the eye, with V. = $\frac{20}{40}$. I saw her about six weeks ago, and this condition had not changed.

DR. A. E. DAVIS, New York: The only further remark that I have to make is that Hirschberg reported a case somewhat similar to that of Dr. Lambert after double cataract extraction. There were retinal hemorrhages in that case, but the retina reattached, with fairly good vision.

ANOMALIES OF THE ACCOMMODATION CLINICALLY CONSIDERED.

ALEXANDER DUANE, M.D.,
New York City.

Considering the amount of attention devoted to refractive errors and their correction, the study of accommodative anomalies has received comparatively little attention from ophthalmologists. Yet those who deal day after day in their consulting-rooms with the problems of refraction work recognize, I am sure, that these anomalies often occasion considerable trouble, so that if we fail to diagnosticate and treat them, we are not doing our whole duty to our patients.

This neglect of an important subject has been due, we believe, not so much to a failure to recognize its importance, as to the fact that hitherto the precise data on which a satisfactory study should be based have been lacking. For it is evident that, before we can say what the symptoms of abnormal accommodation are, we must have a clear notion of what we mean by normal accommodation. In other words, we must, by defining the limits of normal accommodative action, determine when any given accommodation can be called subnormal or supernormal. Up to a few years ago extensive studies on this point were lacking.

This lack the author tried to supply by a series of researches which he has pursued for nine years, and reports of which have been made to this Society in 1908, and to the Section on Ophthalmology of the American Medical Association in 1910 and 1912. Contrary to the impression gathered by some, the main end of these researches was different from that sought by Donders and other predecessors in this field. These observers aimed to obtain a curve representing the

mean accommodation at each age. The author's curves and tables showed this, and, in addition, the maximum and minimum normal accommodation at each age from ten to sixty. As was pointed out in these papers, from these values, which were based on some 12,000* measurements made on 1500 cases, one could determine with fair certainty, as one could not from the values reported by preceding investigators, whether the accommodation in any given case was normal or not, and, if abnormal, how far it varied from the lower or upper normal limits.

The values obtained are shown in the table, page 388.

METHOD OF TESTING ACCOMMODATION.

The way in which this table is used is as follows: The patient whose accommodation we are testing is placed with his back to the light (good, diffuse daylight should be used when possible), and is directed to look sharply at the test-object (a fine black line bisecting a white parallelogram which is mounted on a disc of black velvet). The eye not under examination is covered, and the test-object is brought toward the patient along a Prince's rule until the fine line blurs, and then is carried back and forth several times until we ascertain the nearest point at which the line can be seen with perfect distinctness. The position of this nearest point on the rule indicates the accommodation in diopters.

In order to make the tests comparable with the table the measurement must be made from the anterior focus of the eye, *i. e.*, a point 13 mm. in front of the cornea, or, practically, from the plane of the trial-frame or the patient's glasses. Furthermore, the patient must either be provided with his full refractive correction or, if not, a corresponding allowance must be made in the reading found.

Repeated tests should be taken, and care must be exercised that the results are not vitiated by the patient's inattention

* Confirmed by some 3000 taken since.

TABLE OF NORMAL VALUES OF THE ACCOMMODATIVE POWER AT ALL AGES FROM 8 TO 68.

Accommodation is given in diopters and tenths, near-point being measured from the anterior focus of the eye, *i. e.*, from a point 13 mm. in front of the cornea.

Age	Lower Limit in Normal Cases	Mean Value	Usual Upper Limit	Extreme Upper Limit
8	11.7	13.8	15.4	16.4
9	11.6	13.6	15.2	16.2
10	11.4	13.4	15.0	16.0
11	11.2	13.3	14.9	15.8
12	11.1	13.1	14.7	15.6
13	10.9	12.9	14.5	15.4
14	10.8	12.7	14.3	15.3
15	10.7	12.6	14.1	15.2
16	10.5	12.4	13.9	14.9
17	10.3	12.2	13.7	14.6
18	10.1	11.9	13.5	14.4
19	9.9	11.7	13.2	14.2
20	9.7	11.5	13.0	14.0
21	9.4	11.2	12.8	13.7
22	9.2	10.9	12.6	13.5
23	8.9	10.6	12.3	13.2
24	8.7	10.4	12.1	13.0
25	8.4	10.2	11.8	12.7
26	8.2	9.9	11.6	12.4
27	7.9	9.6	11.3	12.1
28	7.6	9.4	11.1	11.8
29	7.3	9.2	10.7	11.5
30	7.1	8.9	10.4	11.2
31	6.7	8.6	10.2	10.8
32	6.4	8.3	9.9	10.5
33	6.1	8.0	9.6	10.2
34	5.9	7.7	9.2	9.9
35	5.6	7.3	8.9	9.6
36	5.3	7.1	8.6	9.4
37	4.9	6.8	8.2	8.9
38	4.6	6.5	7.9	8.6
39	4.3	6.2	7.6	8.2
40	4.0	5.9	7.2	7.8
41	3.6	5.4	6.8	7.5
42	3.2	5.0	6.4	7.0
43	2.8	4.6	5.9	6.5
44	2.5	4.2	5.5	6.1
45	2.2	3.7	5.2	5.6
46	1.9	3.3	4.8	5.1
47	1.7	2.8	4.3	4.5
48	1.5	2.5	3.9	4.0
49	1.3	2.2	3.4	3.4
50	1.2	2.0	3.0	3.0
51	1.1	1.8	2.6	2.6
52	1.0	1.6	2.3	2.3
53	0.9	1.5	2.1	2.1
54	0.9	1.4	2.0	2.0
55	0.8	1.3	1.9	1.9
56	0.8	1.2	1.8	1.8
57	0.8	1.2	1.7	1.7
58	0.7	1.2	1.7	1.7
59	0.7	1.1	1.6	1.6
60 to 68	0.7	1.1	1.5	1.5

or lack of comprehension of what is expected of him. Care must be taken, too, that we do not consider as due to failure of accommodation blurred vision caused by insufficient illumination, uncorrected errors of refraction, opacities in the media, or retinal disease.* Quite often a very faint and hardly distinguishable central scotoma produces a condition closely simulating an insufficiency of accommodation.

If, now, in any given case, repeated tests, properly made, show that the accommodation lies outside of the normal limits given for a person of the patient's age (see table), we may characterize the finding as abnormal.

PRELIMINARY REPORT ON ABNORMAL ACCOMMODATION.

In the papers cited, and in one read before the New York State Medical Association in 1913,† the general character of the variations from the normal was indicated. The present paper aims to give a more precise description of these anomalies, derived from a study of some 170 cases. These do not include all the cases seen, nor is the study offered as being in any sense complete. It is, in fact, preliminary only.

Varieties of Abnormal Accommodation.—The abnormalities of accommodation that have been observed are as follows:

1. The accommodation may be persistently below the lower normal limit (*insufficiency of accommodation*).

2. Akin to this condition, and, indeed, often constituting one stage of it, is that in which the accommodation is normal in amount, but is sustained only with effort and soon gives out (*difficult or ill-sustained accommodation*).

3. There is difficulty or sluggishness in changing from one accommodative state to another (*inertia of accommodation*).

4. The accommodation in the two eyes is not the same (*inequality of accommodation*).

5. The accommodation lies persistently above the usual normal limit (*excessive accommodation*).

* For precautions to be observed, see the author's papers already cited.

† Jour. New York State Med. Assoc., December, 1913.

Insufficiency of Accommodation.—By insufficiency of accommodation we denote the condition in which repeated tests, made under proper conditions, show that the patient's accommodative power is distinctly and persistently below the normal lower limit for his age (see table). Obviously, also, a patient must be held to have at least a *relative* insufficiency of accommodation if his accommodative power, although above the minimum normal limit for his age, is persistently below his own lower normal limit, as shown by later tests.

Insufficiency of accommodation may be *intermittent* or *constant*, and *transient* or *persistent*. It may also present all grades in intensity. Insufficiency of very high degree (*paresis* and *paralysis* of accommodation) usually differs in origin and character from the moderate insufficiencies, and hence will receive separate consideration. On the other hand, difficult and ill-sustained accommodation differs in no essential way from insufficient accommodation, and will be considered with the latter.

Frequency.—Insufficiency of accommodation is a fairly frequent affection. Precise statistics on this point are not forthcoming as yet, but the opinion may be hazarded that the condition occurs in at least 5 per cent. of our patients.

The following table shows the results as regards sex and age incidence:

Age	Sex			
	Including all Occurring at a Given Age		Excluding those Counted Twice (*i. e.*, Carried on from a Previous Age)	
	Male	Female	Male	Female
10–14	3	5	3	5
15–19	17	5	17	5
20–24	10	12	8	11
25–29	9	6	8	6
30–34	14	6	12	6
35–39	6	14	3	14
40–44	6	16	6	13
45–49	..	6	..	5
	65	70	57	65

The number of cases (122) is too small to warrant sure conclusions, but the following are offered tentatively:

Accommodative insufficiency is not very common before fifteen nor after forty-five. Between fifteen and forty-five it is about as frequent at one age as at another.

From puberty until twenty it occurs three times as often in males as in females.

From twenty to thirty it is about equally frequent in the two sexes.

Between thirty and thirty-five it is about twice as frequent in males. After thirty-five it occurs about three times as often in females as in males.

Types.—Insufficiency of accommodation may theoretically be due either to undue rigidity (accelerated sclerosis) of the lens or to weakness of the ciliary muscle.

We believe, from our observations, that both types actually exist and can usually be distinguished. In the former type, which may properly be called premature presbyopia, and which represents simply a physiologic process carried to an undue degree, the accommodation drops in much the normal fashion from year to year, but in any one year is always below the normal standard. The curve of descent, in other words, parallels the normal curve, but at a lower level.

In the type due to anomalies of ciliary action the insufficiency, being due to pathologic conditions which are themselves variable, varies from time to time, often in a quite haphazard manner, so that the curve of descent exhibits irregular sinuosities and salients. After sinking below normal it may become normal again, which would not be the case in a simple premature presbyopia.

In all probability also mixed cases exist, in which an insufficiency due to impaired ciliary action is added to an insufficiency due to premature sclerosis. These will present the characters of both types.

Associated Muscular Conditions.—It would naturally be

supposed that an insufficiency of accommodation, in which the patient is striving hard to see at near points, would be associated with an undue effort of convergence, *i. e.*, with convergence excess. But this association occurred in only a small minority (about 12 per cent.) of our cases.* Much more frequent was the association with an often marked, but sometimes transient, convergence insufficiency (about 44 per cent. of the cases). In some 10 per cent. other motor anomalies were present, and in about 34 per cent. the exterior muscles were normal.

Etiology.—The causes underlying accelerated sclerosis of the lens, and producing simple premature presbyopia, are, so far as we can tell, unknown.

The causes giving rise to ciliary weakness and causing the second (pathologic) type of accommodative insufficiency have in our cases been as follows:

1. Toxic conditions due to infectious disorders (intestinal toxemia, tuberculosis, influenza, whooping-cough, measles, tonsillar and probably dental infection).

2. Nasal obstruction: this appears to be a frequent cause, especially in the cases associated with convergence insufficiency.

3. Hypopituitarism and other disorders of the internal secretions.

4. Neurasthenia and anemia, brought on by overwork, impaired nutrition, and similar causes. These cases, too, are prominently associated with convergence insufficiency and sometimes with contracted visual fields.

5. Vascular hypertension: it is striking to find how often, in our notes, even in the case of young people, occurs the statement "vessels engorged and tortuous, arteries sharp, discs much injected." With this condition of things or with-

* In these cases of accommodative insufficiency with convergence excess there may be a spasm of accommodation. In other words, the ciliary muscle, weak as it is, may still act in a spasmodic sort of way, just as it does sometimes when partially under the influence of homatropin.

out is found, so often as to indicate its etiologic importance, a persistent, diffuse or reticulate, dense injection of the conjunctiva, with sometimes follicular or other forms of swelling. These cases are specially apt to be associated with considerable burning and smarting from use of the eyes and with intolerance of light. It has seemed to us not improbable that some of the cases with marked irritative symptoms and photophobia might actually be due to the effect of light on the eyes. One case, at least, seemed to be directly brought on by exposure to electric light.

6. Eye-strain, without the other contributing factors, and apart from the injurious effect of light, is a probable but not apparently a very frequent cause.

Symptoms.—The onset may be sudden; even so, apparently, in simple premature presbyopia. In more cases probably the onset is gradual.

The main symptoms are asthenopia—either simple tiring of the eyes or tiring associated with eyeache (sometimes of great severity), with marked irritation and burning of the eyes or with headache; blurred vision, especially for near work; vertigo; aprosexia; photophobia (quite a marked symptom in a number of cases). These symptoms are often due in part to the causal or associated conditions. Thus, the blurred vision at near points and the vertigo are often due to an associated convergence insufficiency or other muscular trouble; the irritative symptoms, to the conjunctival congestion so often present; and the photophobia, to this cause or to the retinal congestion.

An occasional finding, especially in the cases with marked convergence insufficiency, is a concentric contraction of the visual field.

The intensity of the symptoms varies greatly. In many cases they are such as to render eye work almost impossible. On the other hand, especially in simple premature presbyopia, the only complaint may be of impaired vision for near,

which is remedied at once by suitable addition to the glasses for reading.

Course and Duration.—The course and duration naturally depend on the cause. Recurrences may take place, sometimes at long intervals. In simple premature presbyopia the course goes on much as in an ordinary presbyopia, but the necessity for the use of an additional glass for reading begins much earlier—at forty or before.

Treatment.—The treatment is directed to the cause, whenever we can discover the latter. The removal of toxic conditions by remedying an intestinal indigestion or a dental or tonsillar infection; the relief of anemia and malnutrition by the use of tonics and proper diet; the regulation of vascular anomalies; the removal of nasal obstructions; treatment addressed to abnormal glandular conditions—all these are to be employed when required.

Eye-strain naturally should be relieved by proper glasses.

An associated conjunctival injection should be treated with zinc or other astringents.

For the insufficiency itself the following means should be tried:

1. For the blurred vision at near, the distance glass should be supplemented by a proper addition for reading (presbyopic correction for an unnatural presbyopia).

2. Even when the vision is not much blurred, this presbyopic addition may be necessary, especially if there is an associated convergence excess.

3. The accommodation may be stimulated directly by means of reading exercises, or, as I have found useful, by systematic exercise with the fine test-object (line) used in measuring the accommodation. The exercise consists in approximating this to the eye until it blurs, and repeating this manœuver several times, trying each time to bring the line closer and force the eye to accommodate on it. Such exercise can be done two or three times daily.

4. Especially in cases associated with convergence insufficiency it is useful to stimulate both accommodation and convergence by systematic exercise with prisms, bases out, when the eyes are directed at a near object. This is supplemented by practice in converging both eyes on a dot which is brought closer and closer to the eyes until it doubles. These combined exercises are a very considerable help in many cases.

5. The use of pilocarpin as a stimulant to the weak ciliary muscle has not seemed to me of much avail in these cases.

Paralysis of Accommodation.—There is no hard and fast line between paralysis and insufficiency of accommodation. Any insufficiency of considerable amount may be called a paresis, and one that is complete or nearly so, a paralysis. The terms paresis and paralysis are specially applied to marked insufficiencies of sudden development, and either of toxic origin or due to organic lesions of the nervous system. But this is a distinction hard to draw and maintain.

In the great majority of cases paralysis of accommodation is due either to poisons (cycloplegics) or to syphilis and its sequelæ (tabes, general paresis). Paralysis due to these conditions is too well known to require extended description. Of the syphilitic paralyses, it may be said that they are late and very intractable manifestations, of evil prognosis, and yielding little or not at all to treatment, although they may disappear spontaneously and are then not infrequently replaced by paralysis of another kind. They are often unilateral, and are usually, but not always, associated with paralysis of the sphincter iridis.

Other causes that we have seen are:

1. Non-syphilitic disease of the central nervous system (one case). Condition relieved by drainage of cisterna magna, but recurring twice at long intervals.

2. Traumatism (two cases), both associated with paretic mydriasis.

3. Disorders of metabolism in growing boys (two cases, in one associated with almost complete paralysis of convergence.

4. Eye-strain produced by prolonged microscopic work, with voluntary dissociation of the eyes (one case), associated with complete paralysis of convergence.

No diphtherial paralysis was seen, although one would infer, from the text-books, that diphtheria is a frequent cause of accommodative paralysis.

In toxic and traumatic paralyses the lesion is usually peripheral; in syphilitic and parasyphilitic paralyses it is either peripheral, basal, or nuclear. The other types of paralysis are central in origin, and are, therefore, bilateral in character, and are not associated with paretic mydriasis.

A very peculiar picture is afforded by a combined paralysis of accommodation and convergence. In two of my cases the paralysis of both functions was almost total and lasted for months, after which the conditions returned to normal. The symptoms were total inability to use the eyes for anything but distant vision, diplopia increasing *pari passu* as the object looked at approached the eyes, and severe pain in the eyes, with, in one case, also marked headache.

The treatment of these conditions must necessarily be mainly causal.

Unequal Accommodation.—We sometimes find the accommodation unequal in the two eyes. Two reasons may be assigned for this inequality. Either the ciliary muscle may be weaker, or the lens may be more rigid in one eye than in the other. The former sort of inequality occurs quite frequently in ophthalmoplegia interior due to syphilis, tabes, traumatism, or poisons. It can be readily understood that these conditions may affect one eye more than the other, or may affect one to the exclusion of the other. Since in these cases the sphincter iridis and the ciliary muscle are usually affected simultaneously, the inequality in accommodation is

generally associated with inequality of the pupils—the eye with the weaker ciliary muscle having the larger pupil.

When not due to the causes above mentioned, unequal accommodation is usually not associated with inequality of the pupils. In this case, too, it may be caused by unequal action of the ciliary muscle. This was evidently the case in one of our patients, a man of thirty-one, in whom the accommodation in the right eye varied from 3 to 4 D., in the left from 5 to 6, and who also showed marked fatigue of the accommodation in the right eye. The causes here were obscure, but intestinal toxemia and dental infection were present. The symptoms were great asthenopia and a marked spasmodic convergence when he tried to use his eyes for near work.

Most of the other cases of this sort seem to be due to an unequal rigidity of the lenses in the two eyes. In other words, it is a form of presbyopia in which the sclerosis of the lens has advanced faster in one eye than in the other. Like simple premature presbyopia, it is probably a mere physiologic peculiarity, producing no symptoms until the accommodation in one eye or the other has sunk so low as to require the addition of a glass for near work. When this occurs, it is important to take account of the inequality and to determine by experiment whether we shall or shall not make an unequal addition for reading to the distance correction. Patients differ in this regard, some getting on better with such unequal addition, some without.

This condition of unequal presbyopia, or non-pathologic inequality of accommodation, is found mainly in patients between the ages of thirty-five and fifty.

Accommodative Inertia.—Accommodative inertia is the condition in which the patient changes from one accommodative state to another sluggishly or with difficulty. It is analogous to the slow adaptation that some eyes show in passing from light to darkness, or vice versa. It seems some-

times to be a source of real trouble. It is conceivably due to imperfect action of the ciliary muscle (ciliary inflexibility), and if so, might be relieved by accommodative and particularly by converging exercises. But it seems more likely that it indicates advancing sclerosis of the lens, in which case it would be a precursor to a fall in the accommodative power.

Excessive Accommodation.—Excessive accommodation, like insufficiency of accommodation, may be either ciliary or lenticular in origin.

A typical example of excessive accommodation due to ciliary overaction is the accommodative spasm produced by miotics, especially eserin. A similar, less violent action is set up naturally in some patients, causing them persistently to exaggerate a myopia or persistently to reject a hyperopic correction, in spite of continuous wearing. These cases of true spasm of accommodation may occur at any age up to fifty or even beyond. Such spasm may be associated with an actual weakness of accommodative power, and in any case is not an instance of real excess of accommodation.

Lenticular accommodative excess is the condition in which the accommodation is above the usual limit, because the lens is less rigid than ordinary. It is the opposite of premature presbyopia, and may be called delayed presbyopia. Whether, as would seem to be the case, it is always advantageous to have this overplus of accommodation, or whether the overplus may not sometimes indicate a pathologic state,* our observations have not so far enabled us to determine.

DISCUSSION.

DR. S. LEWIS ZIEGLER, Philadelphia: I should like to say a word concerning one phase of the subject discussed by Dr. Duane, namely, subnormal accommodation from obstructive lesions of the nose. I have a collection of these cases, which I hope to publish some day. I found that the

* In one case there were a number of peripheral, punctate, lenticular opacities, rather different in appearance from the ordinary arcus senilis lentis.

most common origin of this disturbance was due to contact-pressure between the middle turbinate and the septum, combined in some cases with a lesion in the sympathetic centers of the septal mucosa. There are other kinds of intranasal pressure that may create the same disturbance, but not so marked. We frequently find a variation in the power of the ciliary muscle. The loss of accommodation may be marked and improve, and then apparently take a second tumble, due entirely to the variable influence of the local lesion in the nose. Occasionally complete recovery occurs.

DR. LUCIEN HOWE, Buffalo, N. Y.: I think if we would take more time and care in examining these cases we would find accommodative insufficiency much more frequently.

I rise, however, to speak of the importance of observing the effect of "minimum" doses of atropin and of eserin. A discussion of this subject can be found in the recent reports made by the Committee on the Study of the Ocular Muscles in the Section on Ophthalmology of the American Medical Association. We know now that about 0.00013 gram is the minimum amount of atropin that will completely relax the accommodation of a normal eye within a given number of minutes. The pharmacists have already manufactured discs containing $\frac{1}{500}$ grain, and by making use of these we have this "minimum" dose which we can place on the conjunctiva. Then, after about twelve to fifteen minutes, the near-point in a normal eye begins to recede; or, if we represent the effect on a system of coördinates, as is usual, we find the curve then begins to fall and later continues to fall in a definite and regular manner, as was shown long ago by Donders. But, on the other hand, when there is insufficiency of accommodation, that curve is different. The fall then is quite rapid, as compared with the normal.

In a similar manner the action of a minimum dose of eserin gives important evidence as to the condition of the accommodation. We know that the minimum dose of this drug for the normal eye is from 0.000005 to 0.00001 gram. If we find that this amount does not contract the pupil within as many minutes as it does normally—in other words, if the curve is much longer than ordinarily—that alone is an indication of accommodative insufficiency.

DR. WILLIAM ZENTMAYER, Philadelphia: I wish to mention that for a long time I was quite skeptical as to the existence of unequal accommodative insufficiency. During a study of the accommodation, however, I found that it was of quite frequent occurrence. Perhaps that is an exaggeration; but it did occur not rarely, and cases that gave a great deal of trouble were relieved by carefully taking the near-point in each eye separately and, where the difference was marked, allowing for it in ordering the glasses.

I fear that our tests for taking the accommodation are not delicate enough to let us know exactly how much to allow. Very often the patient's judgment is poor in regard to the distinctness with which he sees the test-object, and we are led into adding too much or too little.

DR. G. E. DE SCHWEINITZ, Philadelphia: Inequality of accommodation is not of infrequent occurrence, and its determination and correction, in the relief of asthenopia, is a matter of importance.

I should like to confirm the statement of Dr. Duane regarding the frequency with which subnormal accommodation accompanies arteriosclerosis. Writing on the subject some years ago, I referred to certain types of asthenopia which one finds, especially in school-teachers approaching the presbyopic age, in association with high arterial tension. Correction of the vascular hypertension and correction of the subnormal accommodation secure the happiest result. I am satisfied that accommodative power, especially during earlier stages of "old sight," for example, between forty-five and fifty years of age, varies at different times of the day, and I am not satisfied in my determination of the glasses unless opportunity is afforded to compare measurements at different hours on several days. Time does not permit an elaboration of this statement, but the matter is worth investigation.

DR. A. E. DAVIS, New York: There is a certain class of accommodative insufficiency that I should be obliged to have advice as to how to handle; that is, where vision is absolutely perfect for distance, especially the early presbyopes, where there is no convergence insufficiency or excess, where there is orthophoria, and yet the patient is not able to use

the eyes five minutes at a time for reading. I have found this condition in Wall Street people who have been on a nervous tension. In this class, under the best general treatment, so long as they persist in this work, which entails so much nerve strain, I have been unable to give relief for near work. For distance they may have perfect vision, and yet when it comes to using their eyes for near work they cannot persist for as much as five or ten minutes.

As regards unequal accommodative power, I have a patient in whom the inequality amounts to one full diopter, and this correction has been worn for a number of years. The optician, noticing the difference between the two bifocal segments, called me up to be sure that I had not made a mistake. The patient is absolutely comfortable with that marked difference in the reading glasses.

DR. WARD HOLDEN, New York: I should like to ask Dr. Duane how much the maximum difference in the presbyopia of the two eyes may be. I frequently find a quarter of a diopter. There is that difference in my own eyes. I have rarely had occasion, however, to make a difference of more than that in the correction of the two eyes for presbyopia.

DR. ALEXANDER DUANE, New York: In regard to the matter of unequal accommodation in presbyopia, I think that it is one to which we have to pay, and should pay, a great deal of attention. It is not very often that we have to make a difference in the correction, however. Answering Dr. Holden's question, I would say that only occasionally do we find as much difference as a diopter, but we do find it sometimes. A persistent difference between the accommodation of the two eyes is probably due to the fact that one eye has become more presbyopic; that is, lenticular sclerosis has advanced faster in it than in the other. In these cases, even with a difference of 0.50 or 0.75 D., it does not always follow that the patient will accept a corresponding difference in the presbyopic correction. That is simply a matter of trial. Not infrequently, though, I find that I make a difference of a quarter of a diopter with satisfactory results.

One extraordinary case of difference of accommodation in the two eyes was that of the man of thirty-one referred to

in my paper. The accommodation was subnormal in both eyes, but was between one and two diopters more so in one than in the other. In one eye the accommodation was not only weaker than in the other, but was ill sustained. In fact, it would fall one or two diopters during examination. He wore an unequal presbyopic correction with only moderate satisfaction, but that seemed to be the only thing that helped him for the time being. The cause of this condition was unknown, but he had both dental infection and intestinal toxemia, which were in a way to be remedied when I saw him last.

Regarding what Dr. Howe said about the use of minimal doses of atropin and eserin in testing accommodation, I would say that I have had no experience with this. I have made a great many examinations under our usual doses of these drugs to test the effect on accommodation. Under homatropin I have a collection of considerably over 1000 cases that I hope to present the results of. Some of these are extremely surprising, as running counter to the ordinary theories of accommodation presented by Hess (in Graefe-Saemisch) and others.

NERVOUS SYMPTOMS FROM CONTUSIONS OF THE EYE.

EDWARD R. WILLIAMS, M.D.,
Boston, Mass.

The nervous symptoms, including shock, which complicate contusions of the eye, exhibit many interesting variations. My conclusions are based on ten years' observations of 40 cases treated, a few as private patients, the remainder, with few exceptions, cared for at the Boston City Hospital. Since my first report* in 1911 of this kind of ocular injury there has been abundant opportunity to study other interesting cases. As the number of injuries of this character in-

* *Boston Med. and Surg. Journal,* November 9, 1911.

creased it became more and more evident that the most serious complication was some form of shock. Every case of this series had either one of two forms of nervous complications, *e. g.*, surgical shock affecting the patient's entire nervous system, or psychic shock of the eyeball alone. Surgical shock symptoms were usually transitory; the psychic shock of the globe had a marked effect, more or less permanent, on the retina. In order to study all the different phases of these accidents the following conditions will be discussed in detail:

1. Surgical shock at the time of the accident, its relation to cerebral concussion, its exaggeration by psychic influences, as, for instance, the emotions of fear, anxiety, etc.

2. Psychic shock of the eyeball may be divided into two classes: (1) Shock causing reduced vision without permanent damage of the retina; (2) shock effecting serious retinal damage, with permanently reduced vision.

3. Traumatic delirium, often a complication of head injuries, eye operations, etc., and a prominent symptom in two of these cases.

4. Traumatic neuroses, tending to incapacitate the individual from quickly resuming work. Necessity of special after-treatment.

5. Permanent damage to different internal parts of the eye.

In collecting this series of eye contusions great care was taken to consider only those where the globe was struck by rather small objects. It was necessary to keep constantly in mind the confusion of symptoms which might easily come from cerebral concussion. Patients who were struck by baseballs, fist blows, and similar large objects usually suffered from some symptoms of cerebral concussion, and were therefore excluded. Only one patient received a blow hard enough to rupture the globe. As a rule, the object hitting the eye came with just enough force to produce shock, without caus-

ing much permanent damage to the retina. A minority of all the cases showed some varying degree of damage to the interior of the eyeball. Several individuals voluntarily compared the sensations of eye contusions with previous experiences of blows received on the frontal region of the head.

A young man had his eye struck a sharp blow by a tennis-ball while he was playing close to his opponent across the net. He spun around two or three times completely dazed, but did not fall unconscious. The vision was so much blurred that, after some attempt to keep on playing, he was obliged to stop. He was much frightened when, later in the day, he realized that the blurred sight still persisted. He then began to feel weak, nervous, and generally upset. The decrease of vision lasted three days. The physical weakness and nervousness continued throughout the ensuing week, although he tried to attend to his business in a mechanical sort of way. In relating his experience he laid particular stress on the fact that the nervous shock or stunned feeling was quite different from the temporary numbness which he had often experienced during a boxing bout after receiving a severe punch in the face. An examination of his vision two years later showed no reduction from normal and no change in the retina.

This was, therefore, a typical example of psychic shock of the eyeball causing temporary blindness combined with mild but definite shock of the whole nervous system. A second instance showed the nervous symptoms lasting four weeks.

Male, aged forty-two years, was walking in the dark when suddenly the right eye was struck by the end of a wire swinging from a scaffold. He sank to the ground with the agony of the pain. Upon standing up he staggered about, and after five minutes he groped his way, in a very much dazed manner, into a nearby building. He was conscious of great photophobia; the agonizing pain was accompanied by "a sense of roaring and banging from his eye back to the occiput, often sounding like telegraph wires singing in the wind." He felt weak, faint, and chilly. After waiting half an hour

he took the train home. The only relief to the paroxysms of pain was by means of a firm pressure bandage. He then realized he was keyed up to quite a high tension, but found it almost impossible to relax. After going to bed he suffered one severe paroxysm of pain at 2 A. M., but managed to get some sleep undisturbed by dreams.

The eye was examined at my office the following morning, February 10, 1915. The only sign of an external injury was a very small tear of the conjunctiva in the outer lower quadrant. There was also considerable ciliary injection. The pain, for the moment, was not so intense, and was relieved a little by using cocain and dionin. However, the examination was no sooner completed than another paroxysm of pain started. He remarked, "The thumping and banging and singing in my head have returned again." V. R. E. = $\frac{5}{3\frac{1}{2}}$; L. E. = $\frac{5}{6}$.

The second night was more distressing than the first. The pain came in paroxysms, he felt chilly and very restless, even when the pain sometimes became less. It was nearly forty-eight hours before practically all pain ceased. As a result he had a good rest during the third night and felt consequently much stronger and in better spirits. During my second examination he mentioned the great mental depression which, it will be seen, lasted so long during the convalescence. V. R. E. = $\frac{5}{1\frac{1}{6}}$; L. E. = $\frac{5}{8}$. The ciliary injection was considerable, and was accompanied by great tenderness of the globe. The fundus apparently was normal (photophobia made all examinations very distressing).

On February 15th he worked in his office, against my advice, two hours; and later, while at luncheon, he "felt in an apathetic mood and more like sitting quiet with his eyes closed than eating." On the third day the conditions had improved greatly. V. R. E., corrected = $\frac{5}{1\frac{1}{3}}$ + ; L. E., corrected = $\frac{5}{8}$ +. The fundus of the injured eye, using the upright image for the first time, appeared normal.

Eight days later he reported that after getting home he feels like keeping absolutely quiet, and has no desire to make any mental effort. A slight mental depression and irritation at being deprived of his ability to do his regular office work comes back at times. V. R. E. = $\frac{5}{8}$; L. E. = $\frac{5}{8}$ +.

On March 2d V. R. E., corrected = $\frac{5}{8}$ +; L. E. = $\frac{5}{6}$ +.

The ciliary injection was gone, but the eye was weak and easily fatigued during my various tests. He still notices that a lack of ability to see well, and wearing the dark glasses cause a shut-in feeling which is often likely to bring back a depressed mood. At this time he was working about six hours daily, broken by an hour's rest at noon. He notices in the late afternoon he would prefer to sit quiet and not make any physical or mental effort. Upon returning home the right eye aches until he falls asleep.

My last examination, four days later, showed that the eye had regained most of its functions, the corrected vision being $\frac{5}{6}$ +.

The fatigue after a more or less prolonged mental effort, and noticed so often by the patient, was just about the same as the case of a polo player, details of which will be given later. The disposition of each man to work at great tension was also similar. This accounted for many of the exaggerated symptoms of shock.

A third good example was the following:

A workman, aged fifty-two years, seen at the hospital. His right eye was struck squarely by a small bit of wood while working at a lathe revolving 400 times a minute. No signs of abrasions of the skin of the lids seemed to eliminate the confusion which might have come from cerebral concussion. To quote his own words, he "just saw stars, staggered, turned around, and felt confused. Sat down and felt weak and dizzy and wanted to vomit—took away later my appetite upon starting to eat. Have been struck with twelve-inch monkey-wrench falling from a shelf 20 feet onto my head without feeling as many symptoms."

Many others of the hospital cases were workmen inured to hard blows on the head. These men, in spite of robust health, often had shock symptoms quite as pronounced as were observed among the brain workers. Included in this list were two women, neither of whom was of the neurotic type.

Among several boy patients, fright seemed to be a most

important cause of the transient shock. The mental disturbance seemed to last as long as with adults. Among all the cases one fact seemed to stand out very prominently, namely, the disproportionate degree of nervous or shock symptoms to the severity of the blow received. Reference will be made later to the psychic factors which exaggerate shock. Bloodgood explains that sensory impulses sent as the result of an injury or operation are carried to the vital centers of the medulla. The eye, being an organ richly supplied with sensory nerves of the cerebrospinal system, is particularly susceptible to direct blows. Apparently the more richly supplied an organ is with such sensory nerves, the greater the impulse to the brain. In order that the observer be not deceived, he must remember that the eye injury may be only the apparent cause of the mental disturbance. In other words, any serious accident, happening to a neurasthenic or hysteric individual, might cause the same symptom. Slight and apparently trivial blows of the globe frequently cause cerebral symptoms to last several hours at least. If the blow is a hard one, nervous sequelæ may continue during several weeks of a delayed convalescence. Any slight, direct blow on the eye will cause immediately some decrease of vision. This is of great importance, because it is always noted by the patient. As soon as the stunning effect of the blow has subsided somewhat he instinctively compares the sight of both eyes. If he finds a very definite decrease of vision of the injured eye, the horror and fright will frequently upset him still more. This tends to prolong the shock symptoms, which otherwise might be expected to decrease rapidly. The patient cannot possibly know whether permanent damage has been done; the family doctor, if consulted, is not always able to allay his fears. Hence the importance of seeking expert advice and treatment at the earliest possible moment. On the other hand, it must be admitted that many patients ignored the transitory stunning. How-

ever, if this were the case, careful questioning would always give me an interesting and characteristic history of mild shock. The following case history was obtained piecemeal during several visits made by the patient to the hospital. He was at first quite reluctant to describe his sensations before appearing at the hospital for the first treatment:

Waiter, aged twenty-eight years. While stooping over to open a peroxide of hydrogen bottle, the cork flew out and struck his right eye. The bottle was slightly warm, which gave extra force to the blow. Immediately he sank onto his knees, and upon standing up he staggered about the room. He felt cold and shaky, was nauseated and then vomited, and had all the rest of the symptoms of shock. During the night he was very restless from worry and fear of serious consequences to his eye. At the same time slight pain in the eye commenced, becoming intense twenty-four hours after the accident. The next morning he did not work, as he felt unusually weakened. Examination of the injured eye at the clinic showed hyphemia and hemorrhage into the vitreous, which reduced the vision to movements of the hand. During the following week of observation and treatment at the clinic there was very little improvement in the vision of the injured eye. The patient did not return after all hemorrhages were absorbed, so that the end result was unknown.

Naturally, almost all this class of patients were treated in the hospital clinic. In order to avoid missing the interesting details of the accident, we must remember the natural reticence or stupidity of such patients, who are unwilling or unable to give a good description of their sensations.

Several writers have shown interest in the relation of these symptoms of ocular contusions to hysteria. Charles D. Fox* mentions monocular amaurosis as usually due to some trivial trauma which the patient believed capable of producing blindness. Anything which concentrates the attention of the individual upon the sight is sufficiently suggestive to

* Fox, C. D.: The Psychopathology of Hysteria, 1913, p. 90.

make the hysteric person believe the vision is more or less lost. Two of his own cases suffered blindness caused by the patient's concentrated attention upon his eyesight. Gradle* reports monocular amaurosis which followed an injury adjacent to but not involving the affected eye. Coriat† speaks about the increase of nervous symptoms in fatigued or exhausted patients. The emotions will cause most certainly pathologic phenomena during fatigue. An emotional experience also is most likely to recur under states of fatigue.

Usual History of the Accident.—The patient's eye is often hit by the cork flying from a bottle. In addition to the case of the waiter just cited, the following showed the characteristic symptoms of shock and other subsequent peculiar symptoms:

A young woman, a domestic, was attempting to open an ale bottle which had become slightly warmed and the last wire broke and the cork flew against her eye with considerable force. She was unconscious four minutes, and when she was recovered she thought the vision in the injured eye was entirely abolished. She fainted again later in the evening after completing her work. During the night she was very restless, by reason of horrid nightmares in which two men appeared fighting in a boat, using pieces of glass as weapons. This characteristic dream was the subconscious working of the mind referring to her accident. On the following day she made no further complaint of shock symptoms. An examination of her eye showed a characteristic commotio retinæ and hemorrhages extending forward from the retina toward the lens. After two months the trouble cleared away and the vision had improved from $\frac{5}{40}$ to $\frac{5}{5}$.

Other patients have been hit by missiles fired from popguns, by small bits of stone, and by golf-balls. Two men received serious injury from a bung flying out of a cider barrel. One of the most interesting cases in the entire series is as follows:

* Gradle, H.: *Jour. Amer. Med. Assoc.*, April 24, 1909.
† Coriat, I.: Abnormal Psychology, 1914, p. 72.

Man, aged forty-one years, was struck in the right eye by the end of a polo mallet during a game. A very small superficial cut on the upper lid, without any ecchymosis or swelling of either lid, was the best evidence that the blow was received almost entirely on the eyeball. This means that cerebral concussion, as the cause of the nervous symptoms which followed, may be as safely ruled out as in any other cases cited in this series. Since the globe was not ruptured, it means that the ball was tapped at the very end of the mallet's swing, with its greatly decreased impetus. It goes without saying that, had the full strength of the player's stroke fallen on the eyeball, there would have ensued undoubtedly a ruptured globe, fracture of some bones of the orbit, several cuts of the lids, and, lastly, profound, if not fatal, cerebral concussion or compression. As soon as he was hit he fell from his mount, but remained conscious. He vomited, was greatly prostrated, and had all the other well-marked symptoms of shock. His family physician, a very careful and observant man, attended him within half an hour. He told me afterward that all the early shock symptoms were exaggerated and lasted two days. Being called as consultant, my examination was six days after the accident. The injury to the eye was dislocation of the lens, copious hemorrhage into the vitreous, and a slight traumatic retinitis in the macula. At first it seemed best to keep the patient quiet in bed, bromids being given for his nervousness. He became so restless, however, with pent-up emotions it seemed best to allow him to walk outdoors. He became rapidly stronger and much less excitable. He then went off for seven weeks in the Maine forests in the midst of winter. When he returned he looked in splendid physical condition. His ability to use his brain for any serious business was, however, returning rather slowly. He realized he must wait still longer in order to regain his mental balance. My last examination, in December, 1914, was fifteen months after the accident. During the last three months he had worked full time, including three business trips by railroad. In the late afternoon he had a tight feeling of the skull, or sense of mental confusion, or felt slight dizziness. In talking business with other men he then found he could not think clearly, and even casual conversation required a great mental effort. A neuritis of

his left arm, constantly present during the past four years, was made worse by the accident, but showed an encouraging trend toward subsidence. Otherwise this man's physical condition has always been excellent. He always takes plenty of outdoor exercise, and his physical power of resistance is first class. On the other hand, he is temperamentally impatient to any restraint, works in his office at great tension, and is capable of accomplishing a great deal. These characteristics explain the intensity of his shock symptoms and the long-continued neuroses.

Characteristic Shock Symptoms.—The patient, upon being hit on the eye, usually staggers and sinks to the ground, where he remains dazed, but conscious. In several instances the patient loses consciousness several minutes. He groans or cries out in anguish. Severe pain in the globe may come instantly, but sometimes is delayed an hour or more; with severe blows it persists one or several days. The individual feels faint or chilly, looks pale and haggard, is nauseated, and often vomits. Being much weakened, he staggers if he attempts to walk. The pulse is weak and rapid, due to the decrease of blood-pressure. These shock symptoms are usually gone after the first night's sleep. The disturbing dreams occurred in two of these cases. The prostration, both mental and physical, often will last several days.

The subject of surgical shock has been brought again to the attention of all readers by Crile,* in his latest book. In England, Rose and Corless† devote much space to discussing the factors which influence the degree of shock. These same factors are the ones which interest us now while discussing different forms of eye contusion injuries. The authors emphasize the point that the nervous susceptibility of the patient and the expectation or not of the injury are most important factors in the production of shock. If the patient is in a state of tension, expecting some painful sensation, the

* Crile and Lower: Anoci-association, 1914.

† Rose and Corless: Manual of Surgery, 1914.

shock is correspondingly greater. The reverse is also true, that, if the patient's attention is diverted, the shock symptoms are much diminished, though later its effects may be greater. This latter mental attitude was the one almost always seen among my patients. Certainly the unexpectedness of the accident did not prevent a fair proportion of my cases from having well-marked mental disturbances.

An eye injury causing shock was first mentioned by Griesenger* in 1871. A hysteric woman became very melancholic as the result of a trivial injury of the eye by a chip of wood. There were no more details given by the author, the case being noted merely as an exaggerated example of tremendously powerful peripheral irritation which was able to bring on sufficiently characteristic symptoms of a disturbed mental equilibrium.

The above authors describe shock as a depressed condition of the system, associated with a marked fall of blood-pressure resulting from the transmission of some energetic stimulation to the vital centers in the medulla, either from the peripheral, sensory, or sympathetic nerves of an injured part or from the emotional centers.

2. *Psychic Shock of the Eyeball.*—This cause of blindness was carefully studied by von Merz† in 1907, who had many opportunities to treat eye patients during the Russo-Japanese war. The most interesting contusion injuries of the eyes were caused by air concussion following the bursting of shells. He divided his cases into two classes: (A) Psychic shock; (B) a paralytic dilatation of the vessels between the retina and the choroid (Beck's theory).

A. If shock alone is present, there occurs a molecular change in the structure of the retina, but one which is neither macroscopic nor microscopic.

* Griesenger: Pathologie der psych. Krankheiten, 1871.

† von Merz: Schussverletzungen des Auges, *Klin. Monatsblätter für Augenheilk.*, Beilageheft, 1907.

B. If transudation of fluid from the choroidal vessels occurs (even in small amounts), the delicate retinal elements will be more or less destroyed. Ordinarily, there is no ophthalmoscopic change seen. Vision may never be regained, and later an atrophy of the optic nerve will be seen.

An example of Class A was seen at the hospital last autumn.

A young man, well developed and in good health, was fooling with some other men. One of the latter threw, with considerable force, a piece of candle, three inches long, from a distance of ten feet and struck the patient's right eye. He slumped to the ground and remained unconscious four minutes. Upon reviving he felt dizzy, confused, and suffered intense pain in the eye. He was nauseated and felt much weakened. After sitting half an hour he tried to resume his work, but could not do so on account of the great pain, photophobia, and general weakness which lasted throughout the day. The next day, when examined at the hospital, V. R. E. = movements of the hand, on account of the hyphemia; L. E. = normal. Three days later the hyphemia had been absorbed, so that it was possible to examine the fundus. Diagnosis: Neuroretinitis traumatica. The edge of disc blurred; moderate amount of edema of retina near the disc. V. with glass = $\frac{20}{30}$.

The psychic shock of the retina, with the very slight changes seen with the ophthalmoscope, reduced the vision a few days and then disappeared. In such instances there is a hopeful prognosis for a full return of vision. An excellent case, typifying Class B, is the following:

W. R., aged fifteen years, was seen at the Boston City Hospital October 14, 1912. During the previous evening the patient's right eye was hit by a stone the size of a golf-ball, thrown by an older boy 12 feet distant. He was unconscious fifteen minutes; then, upon recovering, began to cry from the severe pain. He felt dizzy and cold. His mother put him quickly to bed and watched him throughout the night. He

cried off and on during the entire night, complaining of the pain. Early the next morning he had nausea, could not eat breakfast, and felt very much "knocked out." Examination of the fundus showed traumatic retinitis, with fresh hemorrhages below the disc. Between this hemorrhage and the edge of the disc was retinal edema, apparently combined with some exudation; the edema was also covering the entire macula region. V. R. E. = $\frac{20}{200}$; L. E. = $\frac{20}{20}$. Nine days later no great change had occurred. V. = $\frac{20}{100}$. The edema, exudation, and blood were absorbing rather slowly. The patient was not seen again.

3. *Traumatic Delirium.*—This condition was observed in two of my cases. The girl having the dream of men fighting with glass was very characteristic. The second instance was as follows:

A man suffered from rupture of the left eye by a blow from a golf-ball. An immediate enucleation of the injured eye was followed, during the succeeding nights, by terrible nightmares. Six times during the first night he seemed to experience over again the accident. If the dreams recur on the following nights, the intensity of the impression gradually fades away. With this patient the dreams recurred during the next ten nights. Restlessness during the first night was spoken of by several patients; the mothers of some of the boy patients observed the same thing.

4. *Traumatic Neuroses.*—This is the mental condition which requires some extra attention. Several of the men already mentioned in this report required weeks and months to regain their proper mental balance. My observation and reëxaminations of these patients have enabled me to come to this conclusion: If the injury is severe enough to have the patient admitted to the hospital, it seems best to me that he shall be kept quietly in bed, at least one day, just as cerebral concussion cases are handled. Worry over the condition of his eye and the doubt in his mind about the final outcome will often be enough to start true traumatic neuroses. An ex-

cellent plea for careful watching of all such accident cases during convalescence has been made by J. J. Thomas.* It seems to apply so aptly to head injuries, and especially to injuries of the eye, that attention is called to it. Those who treat contusions of the eye should realize that a great deal can be done to prevent the development of neuroses by the proper care of the patient during the late convalescence. After the healing of his wounds or after the first shock symptoms have subsided it is not good practice to discharge the patient and tell him he is fit to resume work. The individual does not always feel cured; he is still nervously weakened, and consequently timid of serious complications which may affect the restoration of his full eyesight. The effect of attempting to resume his duties, especially if they are intellectual, is to increase the remaining symptoms. It frequently confirms the fear he has, perhaps unacknowledged to himself, that he never will be able to use his eye and do good mental work again. This fear sets in motion very frequently the chain of symptoms known as traumatic neuroses. The patient should be kept away from too solicitous friends, who, by frequent allusions to his recent accident, tend to keep active a chain of thought which might lead easily to further neuroses. The patient needs repeated assurances that his injured eye is regaining its normal functions. This will do more than any other one thing to restore his confidence.

5. *Permanent Damage to Different Internal Parts of the Eye.*—Varying degrees of serious damage to internal structures of the eye were seen in less than half the total number of cases. Among this group there were, unfortunately, a certain number where the end vision result was unknown. Edema, inflammation, and hemorrhage into the retina, together with rupture of the choroid, were the usual complications, but these pathologic conditions are too frequently seen

* White and Jelliffe: Modern Treatment of Nervous and Mental Diseases, chapter on Traumatic Neuroses, p. 439.

to need further discussion. Two cases only had interesting complications, namely, a dislocation of the lens (already cited in detail) and a well-marked Vossius ring cataract.

DISCUSSION.

Dr. G. S. Derby, Boston, Mass.: It has been my fortune to see quite a number of injuries of the eye produced by its being hit with a ball while the patients were playing various games, such as lawn-tennis, squash rackets, rackets, etc. I can corroborate what Dr. Williams has said in regard to shock. It is certainly surprising to see the instant shock that often takes place when a person is hit in this manner. Some of these persons seem, as they have described it to me, to feel as though hit with a sledge-hammer, the instant shock is apparently so great. This is irrespective of the amount of damage done to the eye, because some of those that I have seen suffered no permanent damage. It does not seem to make very much difference, so far as my experience goes, whether the blow causes little damage to the eye or is sufficient to produce rupture of the eyeball. The instant shock seems often to be the same. As to the later shock, I have not seen it often; but the early shock is very sudden, in my experience, and very great.

Dr. S. C. Maxson, Utica, N. Y.: Before leaving to attend the meeting I saw a patient who had sustained an accident, the shock from which was a surprise to me. The man was injured with the end of a fire-cracker, the stick connected with it striking the lid above the eye. I had to give a poor prognosis, which, perhaps, was unwise. The patient was sent to the hospital, and the head nurse told me that he was pretty nearly in a state of collapse. We had to treat the nervous shock as much as an hour before he recovered and was comfortable.

Since the Employee's Compensation Law has gone into effect one has to be quite sure that a patient is not taking advantage of the law to prolong his convalescence. I am quite discouraged in cases that are up for compensation. In some way they do not get well quickly when the legal question is in abeyance; but when this is settled, I see some very quick restorations.

TRANSACTIONS

of the

American Ophthalmological Society

VOLUME XIV

1915–1916

Philadelphia
American Ophthalmological Society
1916

TRANSACTIONS

of the

American Ophthalmological Society

Fifty-Second Annual Meeting

WASHINGTON, D. C., 1916

VOL. XIV, PART II

Philadelphia
American Ophthalmological Society
1916

PRESS OF WM. F. FELL CO.
PHILADELPHIA

TABLE OF CONTENTS.

	PAGE
List of Officers and Members	421
Necrology	427
Minutes of the Proceedings	437
Three Years' Experience in Sclerocorneal Trephining in Glaucoma: Dr. W. H. Wilmer	453
The Operative Treatment of Partial Staphyloma of the Cornea and of Fistula of the Cornea with a Conjunctival Flap; Conjunctivo-keratoplasty (Kühnt): Dr. Arnold Knapp	466
The Ambulant After-treatment of Cataract Extraction; with a Note on Post-operative Delirium and on Striped Keratitis: Dr. Henry Dickson Bruns	473
Extraction of Cataracts in the Capsule by a Slight Modification of the von Graefe Method: Dr. Ervin Török	482
Improved Capsule Forceps for Intracapsular Cataract Extractions: Dr. F. H. Verhoeff	489
The Etiology of Iritis: Drs. E. V. L. Brown and Ernest E. Irons (by invitation)	495
A Case of Blepharochalasis—A Variety of Dermatolysis: Dr. Robert L. Randolph	518
Observations upon Herpes Corneæ "Febrilis," with Reference, Especially, to Etiology: Dr. Samuel Theobald	520
Two Patients with Unusual Corneal Opacities: Dr. James Bordley, Jr.	527
Ophthalmoscopic Work in the British Base Hospitals: Dr. Allen Greenwood	529
Report of the Removal of a Tumor at the Apex of the Orbit with Preservation of the Eyeball in a Case of Plexiform Neuroma of the Eyelid. Microscopic Examination: Dr. Arnold Knapp	534
An Unusually Large Osteoma of the Frontal, Ethmoidal, and Sphenoidal Sinuses Involving the Orbit and Anterior Cerebral Fossa, Presenting no Subjective Symptoms Other than Proptosis of the Eyeball: Dr. C. A. Veasey	538
Two Cases of Orbital Growth—(A) Mixed Tumor of the Lacrimal Gland. (B) Small Round-cell Sarcoma in an Infant: Dr. T. B. Holloway	542
Recurrent Sarcomata at the Limbus Treated by "Electric Desiccation": Dr. Burton Chance	550
An Epibulbar Melanosarcoma of the Limbus which Completely Disappeared under *X*-ray Treatments: Dr. Albert C. Snell	556

PAGE

Report of a Case of Melanosarcoma of the Orbit Treated with Radium: Dr. Edward B. Heckel 560

A Practical Lamp for Ophthalmic Operations: Dr. A. E. Ewing 565

A Uniform Visual Test-object: Dr. A. E. Ewing 566

A Case of Syphilitic Retinochoroiditis Juxtapapillaris, with Microscopic Examination: Dr. F. H. Verhoeff 568

Tuberculosis of the Retinal Vessels: Dr. Edward Jackson 577

A Clinical and Pathological Study of Two Cases of Miliary Tubercles of the Choroid: Drs. Robert L. Randolph and H. C. Schmeisser (by invitation) 585

Report of Cases of Quinin Amblyopia, with Remarks: Dr. John E. Weeks 593

Retrobulbar Neuritis with Central Scotoma from Toxic Action of Thyroidin: Dr. Myles Standish 608

Optic Neuritis from Syphilitic Leptomeningitis: Dr. William H. Wilder . 614

Cyanosis Retinæ et Conjunctivæ in Connection with Pulmonary Stenosis and Patent Ductus Arteriosus: Dr. Robert Scott Lamb 627

A Case of Pituitary Body Disease with Reference to the Effects of Early Operation: Dr. William Evans Bruner 632

High Hyperopia from a Clinician's Standpoint: Dr. Howard F. Hansell . . 639

Unusual Complications in a Case of Glaucoma after Atropin: Dr. T. Y. Sutphen 645

A Case of Botulism: Dr. Walter B. Lancaster 648

Isolated Paralysis of the Cervical Sympathetic Accompanying Goiter: Dr. H. Maxwell Langdon 661

Paralysis of Divergence: Dr. Ellice M. Alger 665

Divergent Concomitant Strabismus, the Muscular Anomalies and Refractive Errors Accompanying It, and Its Treatment: Dr. Herbert Wright Wootton 678

Color Interlacing and Perimetry: Dr. Clifford B. Walker 684

A Discussion of Some Newer Principles in Dealing with Uncomplicated Cataract: Dr. Derrick T. Vail 718

The Motais Operation for Ptosis: Dr. Francis W. Shine 733

A Study of Hemorrhage into the Anterior Chamber Subsequent to Operations for Hard Cataract: Dr. John M. Wheeler 742

The Etiology of Retinitis Circinata: Dr. Eugene M. Blake 753

The Electromagnet and the Magnet Operation in Ophthalmic Surgery: Dr. John H. Ohly 762

Index to Authors, Vol. XIV 801

Index to Subjects, Vol. XIV 805

American Ophthalmological Society.

FOUNDED 1864.

OFFICERS.

1916–1917.

President,	Dr. Peter A. Callan,	New York, N. Y.
Vice-President,	Dr. W. H. Wilder,	Chicago, Ill.
Secretary-Treasurer,	Dr. Wm. M. Sweet,	Philadelphia, Pa.

MEMBERS.

* Associate Members. O. M. Original Members.

Elected.	Name.	Residence.	Place.
1896	Dr. A. E. Adams,	Balmville, Cor. North,	Newburg, N. Y.
1915	*Dr. Ellice M. Alger,	40 East 41st Street,	New York, N. Y.
1899	Dr. A. N. Alling,	257 Church Street,	New Haven, Conn.
1896	Dr. G. W. Allyn,	Empire Building,	Pittsburgh, Pa.
1882	Dr. A. Alt,	Metropolitan Bldg.,	St. Louis, Mo.
1883	Dr. J. A. Andrews,	1229 State Street,	Santa Barbara, Cal.
1913	Dr. Fred. D. Bailey,	260 Hancock Street,	Brooklyn, N. Y.
1911	Dr. Arthur J. Bedell,	344 State Street,	Albany, N. Y.
1908	Dr. Geo. H. Bell,	40 East 41st Street,	New York, N. Y.
1905	*Dr. Jas. H. Bell,	Moore Building,	San Antonio, Texas.
1916	*Dr. Eugene M. Blake,	55 Trumbull Street,	New Haven, Conn.
1909	Dr. James Bordley, Jr.,	330 N. Charles Street,	Baltimore, Md.
1881	Dr. H. W. Bradford,		Wolfboro, N. H.
1908	Dr. E. V. L. Brown,	122 S. Michigan Blvd.,	Chicago, Ill.
1907	Dr. Wm. E. Bruner,	New England Bldg.,	Cleveland, Ohio.
1901	Dr. H. D. Bruns,	211 Camp Street,	New Orleans, La.
1910	*Dr. E. U. Buckman,	70 S. Franklin Street,	Wilkes-barre, Pa.
1906	Dr. W. K. Butler,	1207 M Street, N. W.,	Washington, D. C.
1906	Dr. W. Gordon M. Byers,	346 Mountain Street,	Montreal, Canada.
1909	Dr. L. W. Callan,	452 Fifth Avenue,	New York, N. Y.
1876	Dr. Peter A. Callan,	452 Fifth Avenue,	New York, N. Y.
1885	Dr. F. P. Capron,	118 Angell Street,	Providence, R. I.
O. M.	Dr. W. H. Carmalt,	261 St. Ronan Street,	New Haven, Conn.
1898	Dr. J. T. Carpenter,	2040 Chestnut Street,	Philadelphia, Pa.
1911	Dr. James J. Carroll,	405 N. Charles Street,	Baltimore, Md.
1904	Dr. Burton Chance,	235 So. 13th Street,	Philadelphia, Pa.

Elected.	Name.	Residence.	Place.
1893	Dr. H. B. Chandler,	34½ Beacon Street,	Boston, Mass.
1904	Dr. J. W. Charles,	509 Humboldt Bldg.,	St. Louis, Mo.
1893	Dr. Wm. Cheatham,	305 W. Chestnut Street,	Louisville, Ky.
1893	Dr. F. E. Cheney,	64 Commonw'th Ave.,	Boston, Mass.
1898	Dr. J. H. Claiborne,	8 West 40th Street,	New York, N. Y.
1912	*Dr. E. W. Clap,	390 Commonw'th Ave.,	Boston, Mass.
1891	Dr. C. F. Clark,	188 East State Street,	Columbus, Ohio.
1903	Dr. Wm. G. Craig,	15 Fuller Building,	Springfield, Mass.
1912	*Dr. G. S. Crampton,	1700 Walnut Street,	Philadelphia, Pa.
1890	Dr. C. M. Culver,	36 Eagle Street,	Albany, N. Y.
1906	Dr. R. J. Curdy,	1134 Rialto Building,	Kansas City, Mo.
1902	Dr. Colman W. Cutler,	24 East 48th Street,	New York, N. Y.
1907	Dr. A. E. Davis,	50 West 37th Street,	New York, N. Y.
1878	Dr. W. S. Dennett,	8 East 49th Street,	New York, N. Y.
1909	Dr. George S. Derby,	7 Hereford Street,	Boston, Mass.
1874	Dr. Lewis S. Dixon,	232 Clarendon Street,	Boston, Mass.
1912	Dr. Oscar Dodd,	30 N. Michigan Blvd.,	Chicago, Ill.
1902	Dr. Alexander Duane,	139 E. 37th Street,	New York, N. Y.
1908	Dr. J. L. Duncan,	Jenkins Bldg.,	Pittsburgh, Pa.
1912	Dr. E. C. Ellett,	Exchange Building,	Memphis, Tenn.
1887	Dr. J. B. Emerson,	40 East 41st Street,	New York, N. Y.
1890	Dr. Arthur E. Ewing,	Metropolitan Bldg.,	St. Louis, Mo.
1896	Dr. T. H. Fenton,	1319 Spruce Street,	Philadelphia, Pa.
1915	*Dr. Carl Fisher,	102 S. Franklin Street,	Rochester, Minn.
1893	Dr. George F. Fiske,	25 E. Washington Street,	Chicago, Ill.
1909	Dr. M. L. Foster,	211 Centre Avenue,	New Rochelle, N. Y.
1904	Dr. W. H. Fox,	1826 Jefferson Place,	Washington, D. C.
1913	*Dr. Lee M. Francis,	636 Delaware Avenue,	Buffalo, N. Y.
1902	Dr. Percy Fridenberg,	38 West 59th Street,	New York, N. Y.
1894	Dr. H. Friedenwald,	1029 Madison Ave.,	Baltimore, Md.
1913	*Dr. H. G. Goldberg,	1925 Chestnut Street,	Philadelphia, Pa.
1895	Dr. G. M. Gould,	215 Atlantic Avenue,	Atlantic City, N. J.
1896	Dr. H. Y. Grant,	Falls View,	Ontario, Canada.
1908	Dr. L. S. Greene,	1624 I Street, N. W.,	Washington, D. C.
1905	Dr. Allen Greenwood,	101 Newbury Street,	Boston, Mass.
1914	*Dr. J. Milton Griscom,	1925 Chestnut St.,	Philadelphia, Pa.
1892	Dr. G. W. Hale,	210 Hitchcock Building,	Nashville, Tenn.
1895	Dr. Wm. D. Hall,	416 Marlborough Street,	Boston, Mass.
1887	Dr. H. F. Hansell,	1528 Walnut Street,	Philadelphia, Pa.
1901	Dr. Herbert Harlan,	516 Cathedral Street,	Baltimore, Md.
1889	Dr. David Harrower,	13 Elm Street,	Worcester, Mass.
1896	Dr. N. D. Harvey,	114 Waterman Street,	Providence, R. I.
1904	Dr. H. H. Haskell,	29 Commonwealth Ave.,	Boston, Mass.
1914	*Dr. Edw. B. Heckel,	Jenkins Building,	Pittsburgh, Pa.
1913	*Dr. Chas. R. Heed,	1402 Spruce Street,	Philadelphia, Pa.

Elected.	Name.	Residence.	Place.
1890	Dr. Neil J. Hepburn,	317 West 23d Street,	New York, N. Y.
1896	Dr. Ward Holden,	8 East 54th Street,	New York, N. Y.
1909	Dr. T. B. Holloway,	1819 Chestnut Street,	Philadelphia, Pa.
1906	Dr. C. R. Holmes,	8 East 8th Street,	Cincinnati, Ohio.
1883	Dr. E. E. Holt,	723 Congress Street,	Portland, Me.
1878	Dr. Lucien Howe,	520 Delaware Avenue,	Buffalo, N. Y.
1891	Dr. D. W. Hunter,	80 West 40th Street,	New York, N. Y.
1898	Dr. J. W. Ingalls,	874 Lafayette Avenue,	Brooklyn, N. Y.
1895	Dr. E. E. Jack,	215 Beacon Street,	Boston, Mass.
1885	Dr. Edward Jackson,	318 Majestic Building,	Denver, Col.
1912	Dr. P. C. Jameson,	139 Montague Street,	Brooklyn, N. Y.
1892	Dr. W. B. Johnson,	170 Broadway,	Paterson, N. J.
1908	Dr. B. R. Kennon,	Taylor Building,	Norfolk, Va.
1901	Dr. H. W. Kilburn,	192 Marlborough Street,	Boston, Mass.
1903	Dr. Arnold Knapp,	10 East 54th Street,	New York, N. Y.
1889	Dr. Carl Koller,	681 Madison Avenue,	New York, N. Y.
1888	Dr. Chas. W. Kollock,	86 Wentworth Street,	Charleston, S. C.
1913	Dr. Robert S. Lamb,	The Cecil,	Washington, D. C.
1891	Dr. W. E. Lambert,	112 East 35th Street,	New York, N. Y.
1902	Dr. W. B. Lancaster,	522 Commonw'lth Ave.,	Boston, Mass.
1912	Dr. H. M. Langdon,	2018 Chestnut Street,	Philadelphia, Pa.
1903	Dr. J. C. Lester,	432 Madison Avenue,	New York, N. Y.
1907	Dr. George F. Libby,	530 Metropolitan Bldg.,	Denver, Col.
1883	Dr. J. A. Lippincott,	Jenkins Building,	Pittsburgh, Pa.
1878	Dr. F. B. Loring,	1420 K Street,	Washington, D. C.
1909	Dr. R. G. Loring,	10 Arlington Street,	Boston, Mass.
1899	Dr. D. B. Lovell,	State Mutual Bldg.,	Worcester, Mass.
1914	*Dr. W. H. Lowell,	101 Newbury Street,	Boston, Mass.
1913	*Dr. A. L. Macleish,	1101 Brockman Bldg.,	Los Angeles, Cal.
1891	Dr. F. W. Marlow,	731 University Block,	Syracuse, N. Y.
1878	Dr. W. V. Marmion,	1211 13th Street, N. W.,	Washington, D. C.
1896	Dr. W. B. Marple,	46 W. 53d Street,	New York, N. Y.
1868	Dr. Arthur Mathewson,	Cosmos Club,	Washington, D. C.
1905	Dr. S. C. Maxson,	235 Genesee Street,	Utica, N. Y.
1907	Dr. C. H. May,	698 Madison Avenue,	New York, N. Y.
1887	Dr. Chas. H. McIlwaine,	320 Belleville Avenue,	Newark, N. J.
1874	Dr. C. S. Merrill,	27 S. Hawk Street,	Albany, N. Y.
1908	Dr. H. S. Miles,	417 State Street,	Bridgeport, Conn.
1883	Dr. J. Lancelot Minor,	Randolph Building,	Memphis, Tenn.
1881	Dr. W. F. Mittendorf,	399 Park Avenue,	New York, N. Y.
1916	*Dr. John H. Ohly,	22 Schermerhorn St.,	Brooklyn, N. Y.
1887	Dr. H. S. Oppenheimer,	276 Madison Avenue,	New York, N. Y.
1908	Dr. W. R. Parker,	David Whitney Bldg.,	Detroit, Mich.
1908	Dr. S. McA. Payne,	542 Fifth Avenue,	New York, N. Y.
1895	Dr. R. J. Phillips,	123 So. 39th Street,	Philadelphia, Pa.

Elected.	Name.	Residence.	Place.
1869	Dr. Thomas R. Pooley,	26 Trinity Street,	Newton, N. J.
1898	Dr. W. Campbell Posey,	2049 Chestnut Street,	Philadelphia, Pa.
1895	Dr. H. R. Price,	435 Clinton Avenue,	Brooklyn, N. Y.
1906	Dr. Brown Pusey,	7 West Madison Street,	Chicago, Ill.
1902	Dr. Walter L. Pyle,	1931 Chestnut Street,	Philadelphia, Pa.
1901	Dr. Alex. Quackenboss,	143 Newbury Street,	Boston, Mass.
1906	Dr. McCluney Radcliffe,	1906 Chestnut Street,	Philadelphia, Pa.
1885	Dr. Alex'r Randall,	1717 Locust Street,	Philadelphia, Pa.
1892	Dr. R. L. Randolph,	609 Park Avenue,	Baltimore, Md.
1891	Dr. J. M. Ray,	Atherton Bldg.,	Louisville, Ky.
1901	Dr. R. G. Reese,	50 West 52nd Street,	New York, N. Y.
1889	Dr. R. A. Reeve,	48 Bloor Street East,	Toronto, Canada.
1899	Dr. H. O. Reik,	506 Cathedral Street,	Baltimore, Md.
1912	Dr. W. G. Reynolds,	1165 Dean Street,	Brooklyn, N. Y.
1884	Dr. Stephen O. Richey,	732 17th Street,	Washington, D. C.
1901	Dr. G. O. Ring,	2014 Chestnut Street,	Philadelphia, Pa.
1895	Dr. H. W. Ring,	185 Church Street,	New Haven, Conn.
1908	Dr. J. Norman Risley,	36 7th Street,	New Bedford, Mass.
1880	Dr. Samuel D. Risley,	2018 Chestnut Street,	Philadelphia, Pa.
1900	Dr. Wm. K. Rogers,	188 East State Street,	Columbus, Ohio.
1899	Dr. Dunbar Roy,	Gr. Opera House Blk.,	Atlanta, Ga.
1875	Dr. John D. Rushmore,	129 Montague Street,	Brooklyn, N. Y.
1880	Dr. Robert Sattler,	30, The Groton,	Cincinnati, Ohio.
1912	*Dr. A. C. Sautter,	1421 Locust Street,	Philadelphia, Pa.
1898	Dr. T. B. Schneideman,	1831 Chestnut Street,	Philadelphia, Pa.
1889	Dr. G. E. de Schweinitz,	1705 Walnut Street,	Philadelphia, Pa.
1895	Dr. P. N. K. Schwenk,	1417 North Broad St.,	Philadelphia, Pa.
1913	*Dr. C. E. G. Shannon,	1633 Spruce Street,	Philadelphia, Pa.
1905	Dr. John R. Shannon,	17 East 38th Street,	New York, N. Y.
1906	Dr. H. G. Sherman,	736 Rose Building,	Cleveland, Ohio.
1916	*Dr. Francis W. Shine,	37 East 30th Street,	New York, N. Y.
1911	Dr. J. F. Shoemaker,	1006 Carleton Bldg.,	St. Louis, Mo.
1902	Dr. Wm. A. Shoemaker,	1006 Carleton Bldg.,	St. Louis, Mo.
1902	Dr. Wm. T. Shoemaker,	109 South 20th Street,	Philadelphia, Pa.
1905	Dr. E. A. Shumway,	2007 Chestnut Street,	Philadelphia, Pa.
1912	Dr. E. T. Smith,	Conn. Mutual Bldg.,	Hartford, Conn.
1910	Dr. Albert C. Snell,	53 S. Fitzhugh St.,	Rochester, N. Y.
1909	Dr. F. M. Spalding,	39 Huntington Ave.,	Boston, Mass.
1878	Dr. J. A. Spalding,	627 Congress Street,	Portland, Me.
1911	*Dr. Chas. N. Spratt,	900 Nicollet Avenue,	Minneapolis, Minn.
1884	Dr. Myles Standish,	72 St. James Avenue,	Boston, Mass.
1873	Dr. George T. Stevens,	40 East 41st Street,	New York, N. Y.
1903	Dr. Edw. Stieren,	Westinghouse Bldg.,	Pittsburgh, Pa.
1887	Dr. T. Y. Sutphen,	992 Broad Street,	Newark, N. J.

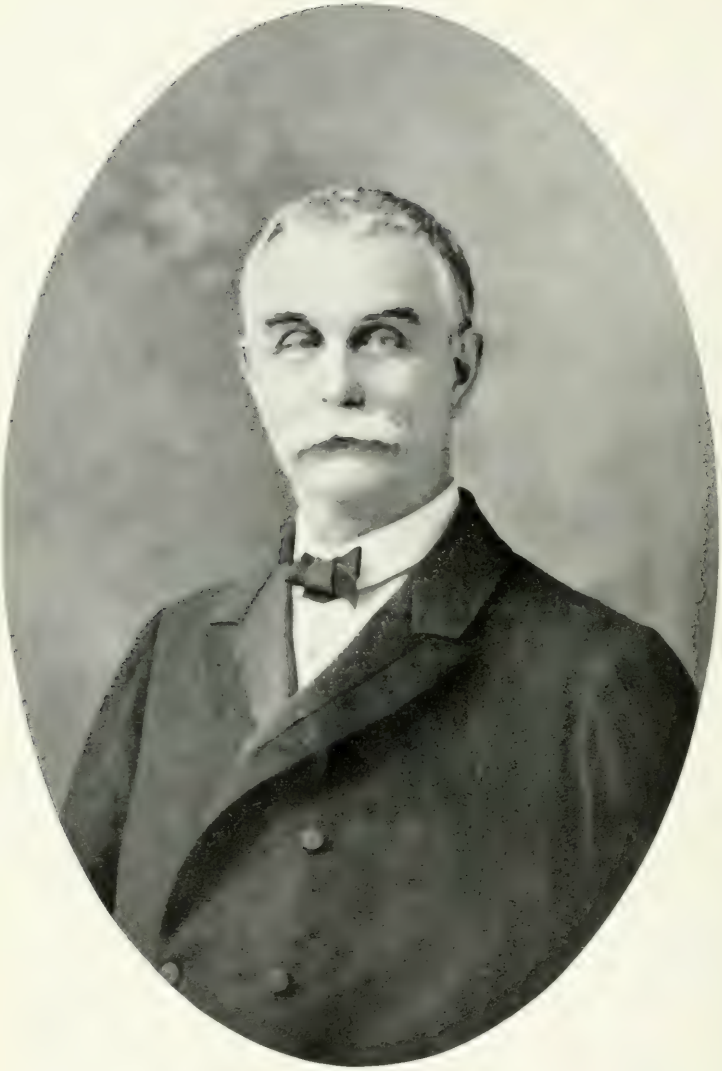
Elected.	Name.	Residence.	Place.
1900	Dr. Wm. M. Sweet,	1205 Spruce Street,	Philadelphia, Pa.
1888	Dr. Lewis H. Taylor,	83 So. Franklin Street,	Wilkes-barre, Pa.
1880	Dr. Samuel Theobald,	Cathedral & How'd Sts.,	Baltimore, Md.
1893	Dr. Ch. Hermon Thomas,	3634 Chestnut Street,	Philadelphia, Pa.
1898	Dr. A. G. Thomson,	Stock Exchange Bldg.,	Philadelphia, Pa.
1903	Dr. Edgar S. Thomson,	405 Park Avenue,	New York, N. Y.
1898	Dr. Jas. Thorington,	2031 Chestnut Street,	Philadelphia, Pa.
1908	Dr. Andrew Timberman,	112 East Broad Street,	Columbus, Ohio.
1911	Dr. Fred. Tooke,	368 Mountain Street,	Montreal, Canada.
1912	*Dr. E. Török,	8 East 54th Street,	New York, N. Y.
1906	Dr. H. H. Tyson,	47 West 51st Street,	New York, N. Y.
1916	*Dr. Derrick T. Vail,	24 East 8th Street,	Cincinnati, Ohio.
1898	Dr. C. A. Veasey,	Paulsen Building,	Spokane, Wash.
1905	Dr. F. H. Verhoeff,	101 Newbury Street,	Boston, Mass.
1903	Dr. F. L. Waite,	68 Pratt Street,	Hartford, Conn.
1916	*Dr. Clifford B. Walker,	697 Huntington Avenue,	Boston, Mass.
1873	Dr. David Webster,	24 East 48th Street,	New York, N. Y.
1893	Dr. J. E. Weeks,	46 East 57th Street,	New York, N. Y.
1914	*Dr. Walter B. Weidler,	62 West 58th Street,	New York, N. Y.
1900	Dr. C. D. Wescott,	22 E. Washington Street,	Chicago, Ill.
1916	*Dr. John M. Wheeler,	80 West 40th Street,	New York, N. Y.
1891	Dr. Edward Wheelock,	26 Gibbs Street,	Rochester, N. Y.
1892	Dr. J. A. White,	200 E. Franklin Street,	Richmond, Va.
1895	Dr. W. H. Wilder,	1417 Peoples Gas Bldg.,	Chicago, Ill.
1910	Dr. Carl Williams,	School L. & Greene St.,	Philadelphia, Pa.
1874	Dr. Chas. H. Williams,	1069 Boylston Street,	Boston, Mass.
1912	Dr. Edw. R. Williams,	1069 Boylston Street,	Boston, Mass.
1893	Dr. Wm. H. Wilmer,	1610 I Street, N. W.,	Washington, D. C.
1888	Dr. F. M. Wilson,	834 Myrtle Avenue,	Bridgeport, Conn.
1906	Dr. J. Scott Wood,	172 Sixth Avenue,	Brooklyn, N. Y.
1900	Dr. Hiram Woods,	842 Park Avenue,	Baltimore, Md.
1915	*Dr. H. W. Wootton,	319 Lexington Avenue,	New York, N. Y.
1881	Dr. J. P. Worrell,	20 South 7th Street,	Terre Haute, Ind.
1905	Dr. Wm. Zentmayer,	1819 Spruce Street,	Philadelphia, Pa.
1903	Dr. S. Lewis Ziegler,	1625 Walnut Street,	Philadelphia, Pa.

Total 195

HONORARY MEMBERS.

1868	Dr. J. S. Prout,	152 Putnam Avenue,	Brooklyn, N. Y.
O. M.	Dr. F. P. Sprague,	229 Commonw'lth Ave.,	Boston, Mass.

Whole number . . . 197



Dr. Benjamin Joy Jeffries.

NECROLOGY.

DR. BENJAMIN JOY JEFFRIES.

FREDERICK E. CHENEY, M.D.,
Boston.

Dr. Benjamin Joy Jeffries, one of the original members of the American Ophthalmological Society, died November 21, 1915, in his eighty-third year. He was born in Boston, March 26, 1833, the son of Dr. John Jeffries, a distinguished physician of his generation and a grandson of an earlier Dr. John Jeffries, a surgeon of the British Army in America during the American Revolution.

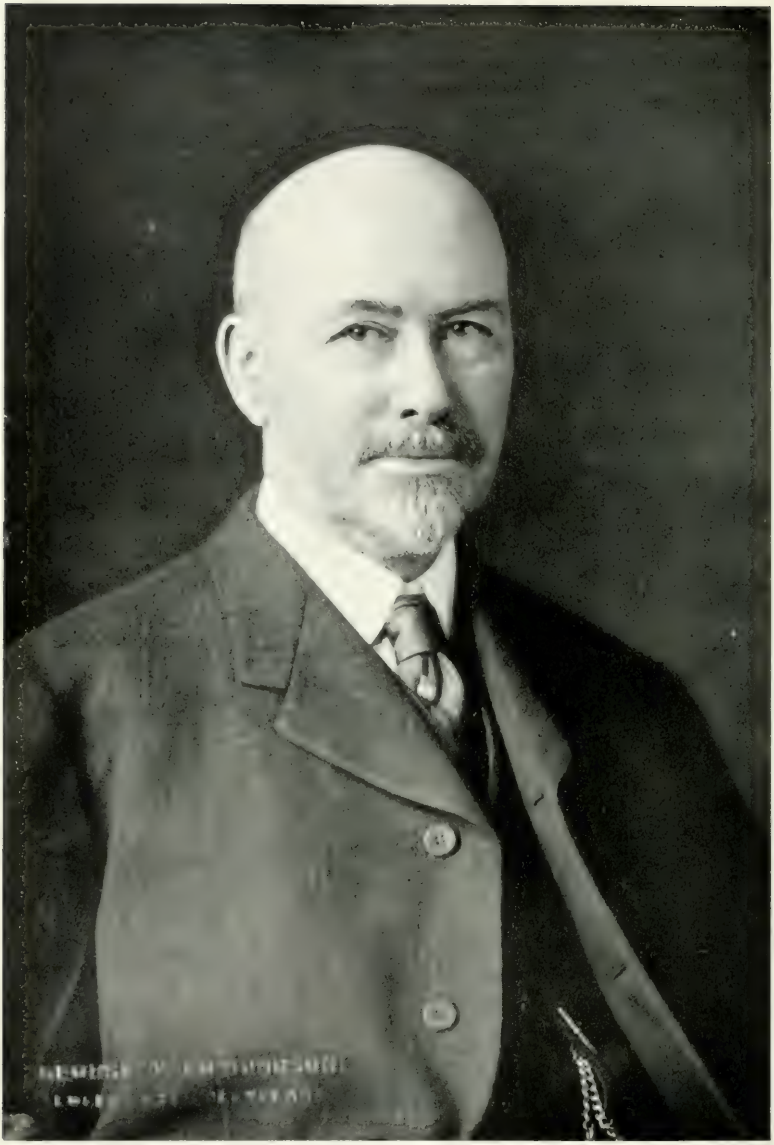
From the Boston Latin School Dr. Jeffries entered Harvard College, receiving the degree of Bachelor of Arts in 1854, and, three years later, the degree of Doctor of Medicine from the Medical Department of the same university. His medical studies were later continued in Vienna, principally under Professor Arlt and Professor Hebra. Returning to Boston, he began practice, confining his work to the two specialties of dermatology and ophthalmology. In connection with Dr. Francis P. Sprague and the late Dr. James C. White, he opened a free dispensary on Eliot Street for the treatment of these two classes of disease. Gradually he gave up the practice of dermatology, devoting his entire time to ophthalmology. In 1866 he was appointed Ophthalmic Surgeon to the Massachusetts Charitable Eye and Ear Infirmary, and continued in active practice at this institution for thirty-six years. Resigning in 1902, he became one of the consulting surgeons, a position he retained up to the time of his death.

As a practitioner of ophthalmology it may be said of Dr. Jeffries that he was extremely diligent and conscientious, faithful and devoted to his patients, and an excellent operator. In his early life he was particularly interested in the proper administration of ether in ophthalmic surgery, and made a special trip to England to demonstrate the superiority of American methods of anesthesia to his English colleagues. A paper, "The Reintroduction of Ether into England," was published in the *Boston Medical and Surgical Journal* soon after his return in 1872.

Dr. Jeffries gained especial prominence and distinction from his work on color-blindness, and will be best remembered for his painstaking and conscientious accomplishment in this branch of ophthalmology. His first paper on this subject, "The Incurability of Congenital Color-Blindness," was published in the *Boston Medical and Surgical Journal* in 1878. This was followed by four other papers during the same year, and in the following year his well-known book, "Color-Blindness, its Dangers and Detection," was issued. Numerous other papers were published in the years that followed, not only in this country, but abroad. Among the more important of these may be mentioned: "Observations on a Peculiar Expression of the Eyes of the Color-Blind" (Trans. International Medical Congress, London, 1881). "Color-Names, Color-Blindness and the Education of the Color-Sense in our Schools" (Education, 1882), and "Report of Examination of 27,927 School Children for Color-Blindness" (School Document No. 13, 1889). His last contribution was, if I am not mistaken, a "Report on Worsteds for Holmgren's Test," read before this Society and published in the Transactions in 1895.

It would be difficult to estimate the practical value of his work during these years, especially in its relation to the problem of railroad transportation and travel by sea.

Dr. Jeffries was descended from a distinguished New



Dr. Benjamin L. Millikin.

England ancestry, and his love and pride in family, with, however, no taint of snobbishness, and his keen interest in the association of its various members with prominent people and events of the past, soon became apparent to any one who was at all intimately associated with him. His house on Chestnut Street, where his father had lived and practised before him, was filled with interesting things of past generations: many that came to him by inheritance and others that were added because of their association with historical people and events. An invitation to visit Dr. Jeffries in his home was always a source of gratification to the younger men of the profession, and his keen and simple enjoyment in displaying and talking of his possessions added greatly to the pleasure of his guest. The remains of the balloon that first crossed the English channel from France with his grandfather as a passenger was always of especial interest to the visitor, as the importance of the event in early aëronautics is, of course, historic.

Dr. Jeffries is survived by one daughter, the wife of Dr. James Howard Means, of Boston. His only son, Charles Shimmin, died in early youth.

Dr. Jeffries was almost the last of his medical generation in Boston. He was a man of kindly and genial disposition, of a distinct and strong personality, and attained an honorable and enviable position in his profession.

DR. BENJAMIN L. MILLIKIN.

WILLIAM EVANS BRUNER, M.D.,
Cleveland, Ohio.

In the death of Dr. Benjamin L. Millikin, the American Ophthalmological Society has lost another of its old and well-known members. Dr. Millikin was descended, on both his

father's and mother's side, from Emanuel Millikin, who, with his four sons, all born in southern Scotland, fought with William of Orange at the Battle of the Boyne in 1690, and for their services was given land in Kilnumery, County Leitrim, Ireland. His father, Christopher, born in 1805, helped to "break" the wilderness in northern Ohio.

The subject of this sketch, the fifth of six children, was born on a farm near Warren, Ohio, December 24, 1851. His boyhood was spent upon the farm, and in the little country school-house, under difficulties little now appreciated, he secured his early education. Later he attended school at Warren, often walking the distance through all kinds of weather, while in the summer he worked on the farm.

After graduation from Allegheny College, Meadville, Pa., in 1874, he entered the Medical Department of the University of Pennsylvania, from which institution he was graduated in 1879, and then pursued post-graduate courses abroad. Returning to this country he served as a resident physician in the University of Pennsylvania Hospital; later, in the Philadelphia Children's Hospital; and finally, in the Wills Eye Hospital.

Following this he settled in Cleveland, but for a short while also maintained an office in his home town of Warren. The rapid growth in his practice soon made it necessary for him to give up his Warren office and devote his whole time to his practice in Cleveland, where he has held an eminent position in ophthalmology from that time until his death.

In 1883 he became Professor of Diseases of the Eye in the Medical Department of Western Reserve University, and continued actively in this position until 1912, when he became Professor Emeritus. He was appointed visiting ophthalmologist to St. Vincent's (Charity) Hospital in 1884, and from 1892 to 1912 was visiting ophthalmologist to Lakeside Hospital, and afterward, until the close of his life, was senior ophthalmologist.

In 1900 he became dean and executive officer of the medical faculty of Western Reserve University, serving in this capacity until 1912. He held this position during an important period in the development of the medical school, during which time the standard of scholarship in the school was raised so that the entrance to the medical course depended upon college graduation. He also saw the endowment of the school increased tenfold during his twelve years of service. He was not only an earnest supporter of the increased requirements for admission, and of the lengthening of the course, but ever strove for the maintenance of a high standard of excellence in the work of the institution throughout the course.

His interest in medical education was shown, not only in his work in the college, but also in his active support of the Cleveland Medical Library Association. He was president of the Library for two years, giving it much of his time and the benefit of his judgment and experience. During his last illness he was constantly planning for its future growth and development. He had just been reëlected as its executive head a short time before his death.

He was married in 1891 to Miss Julia W. Severance, who, with five children, two sons and three daughters, survives him.

Dr. Millikin's health had been poor for ten years, but, ever uncomplaining, he made no mention of it, and the end, though following a short illness not in itself thought serious, came very suddenly, and was a great shock to his many friends.

Reared on the farm, he was ever very fond of nature, and familiar with it. He found great pleasure in taking rambles with his children through the fields and woods, and would evince disgust when they could not recognize a tree by its bark or leaf or know a bird by its song. His home was just outside the city, and he found great delight in the develop-

ment of its gardens and surroundings, and great interest also in the growth of the surrounding country.

He was gentle in his disposition, quiet and affable in his manner, with a marked degree of self-control. While he was a man of decided opinions, he never sought to force them upon other persons. He was ever thoughtful of others and considerate and charitable toward them and their opinions. He was modest, and, avoiding publicity himself, he heartily disliked the self-advertising so prevalent today among some physicians. His professional career was marked throughout by devotion to high ideals. While he was conservative to a marked degree, his judgment was excellent, and his advice highly valued by consulting physicians as well as his patients. He was only a moderate contributor to medical literature—to various journals and the Transactions of this Society. He also wrote the article upon "Diseases of the Eyelids" in the American Text-Book of Diseases of the Eye, Ear, Nose, and Throat.

DR. CHARLES HEADY BEARD.

WILLIAM H. WILDER, M.D.,
Chicago.

Dr. Charles Heady Beard came of a family who were among the early settlers of Kentucky, and one of the oldest towns in that State, Bardstown, the county seat of Nelson county, was originally named Beard's town, from his ancestors who settled there.

He was born at Elk Creek, Spencer County, January 27, 1855, son of James P. Beard, a farmer of that community, and Emarine Heady Beard, who was a daughter of Dr. J. J. Heady, a pioneer physician of that county.

His preliminary education was received in the public

Dr. Charles Heady Beard.

schools of Spencer county and in the Transylvania University of Lexington, Kentucky.

He is reported to have been a bright, precocious boy, fond of books, and with much artistic talent, which in early youth manifested itself in a great fondness for drawing, and he painted a number of creditable pieces of canvas. At the age of fourteen years, as a result of a fall from a threshing machine, it became necessary to amputate one foot above the ankle, and this severe accident probably had a marked influence upon his subsequent career. Much of his early boyhood and youth was spent with his grandfather, Dr. Heady, under whose inspiration and guidance he began the study of medicine. This study was continued in the medical department of the University of Louisville, from which institution he took his Doctorate in Medicine in 1877.

He removed to Cannelton, Indiana, where he began the practice of general medicine, and met with success. After several years he decided to abandon general practice, chiefly because of his physical defect, and to devote himself to a specialty. Accordingly in 1883 he went to New York, and after courses of study at the Post-graduate Medical School, the Polyclinic, and Ophthalmic and Aural Institute, he secured the position of house surgeon in the Manhattan Eye and Ear Hospital, entering upon his duties May 1, 1884, and serving one year. During this term of service he began his collection of ophthalmoscopic drawings to which he constantly added during life. He was rated as unusually clever and enjoyed a reputation with the visiting surgeons for good judgment and skill. The late Dr. Cornelius R. Agnew, at that time Executive Surgeon at the Manhattan Hospital, repeatedly expressed admiration for Dr. Beard and his work.

At the end of his hospital year he went abroad to continue his studies in London, Vienna, Zurich, and Paris, spending most of his time in Paris, where he became acquainted and

associated with Panas and other members of the French school of ophthalmology, of which he was a great admirer.

On his return to this country he took charge of the practice of Dr. F. M. Wilson, of Bridgeport, Conn., during the latter's absence in Europe, and then in the latter part of 1886 he located in Chicago, where he began his practice of ophthalmology and otology. In 1887 he became assistant surgeon in the Illinois Eye and Ear Infirmary, and surgeon in 1890, a position he occupied until his resignation a few months before his death. He was also for many years ophthalmologist to the Passavant Memorial Hospital. He was interested in the general and special medical societies of Chicago and Illinois, and was an active member of several of them. He was one of the charter members of the Chicago Ophthalmological Society, to the meetings of which he made valuable contributions, and in 1903 was elected its President.

He was elected a member of the American Ophthalmological Society in 1907. He was also a member of the American Medical Association, the American Academy of Ophthalmology and Oto-laryngology, and the American College of Surgeons.

He was a careful student of ophthalmic literature as evinced by his own writings, which, although not numerous, were valuable contributions to the subject considered. In 1910 appeared his *Ophthalmic Surgery*, which was received as a valuable guide and which expressed so much of the author's ingenuity and skill as an operator. He contributed an important chapter to the American System of Ophthalmic Operations on Blepharoplasty, and in 1913 a volume to the International System of Ophthalmic Practice, entitled "Ophthalmic Semiology and Diagnosis," illustrated with many of his own beautiful drawings in black and white and colors.

His literary style was clear and concise, and he frequently embellished his sentence with some quaint allusion or figure that made it peculiarly forceful.

As a clinician he was thorough, conscientious, and of rare judgment, and these traits of character naturally attracted to his private office a large clientele. His quiet, firm manner inspired confidence and admiration in his patients. As an operator he was ingenious and resourceful, with a good technic. He took pleasure in plastic work, in which he excelled, and found expression of his artistic talent. His early fondness for drawing and painting later developed into a love for portraying the multitudinous changes that find expression in the background of the eye. His wonderful drawings of the fundus oculi, with such conscientious regard for the smallest details, have been admired by all who have seen them. It is doubtful if he was ever surpassed, and it is certain that he was rarely equaled by any other individual in this line of artistic work. It is to be hoped that these valuable drawings will be carefully preserved and at some future time reproduced for the benefit of ophthalmic science. Unfortunately this task which he had set for himself was not completed.

Dr. Beard was a man of genial character who made many friends and held them. He had a quiet demeanor, and although firm was rarely contentious. His fortitude was shown in his final illness. About eighteen months before his death, because of some ill-defined symptoms not previously noticed, he consulted a physician who discovered a suspicious mass in the upper part of the abdomen. An exploratory operation revealed a malignant condition of the abdominal viscera that was inoperable. With his doom sealed, he went about his work and among his friends with the calm courage of a stoic and did not cease his task until about three weeks before the end. He died January 3, 1916.

Dr. Beard married in 1888 Laura Clark, of Cannelton, Indiana, and she and two children, Hallard and Charlotte, survive him. His son Hallard is a student in Rush Medical College.

MINUTES OF THE PROCEEDINGS.

FIFTY-SECOND ANNUAL MEETING.

WASHINGTON, D. C., *May 9, 1916.*

The Fifty-second Annual Meeting of the Society was called to order at 9 A. M. by the President, Dr. George E. de Schweinitz, of Philadelphia.

The following Committee was appointed:

Auditing Committee: DR. G. ORAM RING.

The scientific program was then taken up, and the following papers were read and discussed:

1. "Three Years' Experience in Sclerocorneal Trephining in Glaucoma." W. H. Wilmer, M.D.

Discussed by Drs. Marple, Bruns, Lambert, Ewing, Timberman and de Schweinitz.

2. "The Operative Treatment of Partial Staphyloma of the Cornea and of Fistula of the Cornea with Conjunctival Flap; Conjunctivo-keratoplasty (Kuhnt)." Arnold Knapp, M.D.

Discussed by Drs. Roy and Weeks.

3. "On the Ambulant After-treatment of Cataract Extraction; with a Note on Post-operative Delirium and on Striped Keratitis." Henry Dickson Bruns, M.D.

Discussed by Drs. Knapp, May, Weeks, Ellett, Reeve and Bruns.

4. "Extraction of Cataracts in the Capsule by a Slight Modification of the von Graefe Method." Ervin Török, M.D.

5. "An Improved Capsule Forceps for Intracapsular Cataract Extractions." F. H. Verhoeff, M.D.

Discussed by Drs. Fisher and Lancaster.

6. "The Etiology of Iritis." E. V. L. Brown, M.D., and Ernest E. Irons, M.D. (by invitation).

Discussed by Drs. de Schweinitz, Pyle, Marple, Weeks, Theobald, H. H. Briggs, Foster, Brown and Irons.

7. "A Case of Blepharochalasis—A Variety of Dermatolysis." Robert L. Randolph, M.D.

Discussed by Drs. Stieren and Heckel.

8. "Observations upon Herpes Corneæ 'Febrilis,' with Reference, Especially, to Etiology." Samuel Theobald, M.D.

Discussed by Drs. Verhoeff and Theobald.

9. "Two Patients with Unusual Corneal Opacities." James Bordley, Jr., M.D.

10. "Ophthalmoscopic Work in the British Base Hospitals." Allen Greenwood, M.D.

Discussed by Drs. Hansell, Risley, Lambert, and Greenwood.

The following physicians were introduced as guests of the Society, and invited to take part in the discussions: Drs. F. C. Todd, Minneapolis, Minn.; Ernest E. Irons, Chicago; Dorland Smith, Bridgeport, Conn.; John W. Burke and E. L. Morrison, Washington, D. C.; H. C. Schmeisser, C. A. Clapp, E. H. Rowe and Alan Woods, Baltimore, Md.; Wendell Reber and M. Buchanan, Philadelphia; W. S. Frost, Spokane, Washington; L. A. Nelson, St. Paul, Minn.; E. E. Rosenow, Rochester, Minn.; W. W. Blair, Pittsburgh, Pa.; W. W. Pearson, Des Moines, Iowa; Thomas O. Edgar, Dixon, Ill.; J. W. Jervey, Greenville, S. C.; H. H. Briggs, Asheville, N. C.; C. B. Wylie, Morgantown, W. Va.; and Major Theodore Lyster, U. S. Army, Washington, D. C.

AFTERNOON SESSION, MAY 9TH.

The meeting was called to order by the President at 4 o'clock. The following papers were read:

11. "Report of the Removal of a Tumor at the Apex of

the Orbit with Preservation of the Eyeball in a Case of Plexiform Neuroma of the Eyelid. Microscopic Examination." Arnold Knapp, M.D.

Discussed by Drs. Ellett and Knapp.

12. "An Unusually Large Osteoma of the Frontal, Ethmoidal and Sphenoidal Sinuses Involving the Orbit and Anterior Cerebral Fossa, Presenting no Subjective Symptoms Other Than Proptosis of the Eyeball." C. A. Veasey, M.D.

Discussed by Drs. Reeve and Jackson.

13. "Two Cases of Orbital Growth. (A) Mixed Tumor of the Lacrimal Gland. (B) Small Round-cell Sarcoma in an Infant." T. B. Holloway, M.D.

14. "Recurrent Sarcomata at the Limbus Treated by Electrical Desiccation." Burton Chance, M.D.

Discussed by Drs. Derby and Chance.

15. "An Epibulbar Melano-sarcoma of the Limbus which Completely Disappeared under *x*-Ray Treatments." Albert C. Snell, M.D.

16. "Report of a Case of Melano-sarcoma of the Orbit Treated with Radium." Edward B. Heckel, M.D.

Discussed by Drs. Zentmayer and Lambert.

17. "A Case of Syphilitic Retino-choroiditis Juxtapapillaris, with Microscopic Examination." F. H. Verhoeff, M.D.

Discussed by Drs. Holloway, Zentmayer, de Schweinitz, and Verhoeff.

EXECUTIVE SESSION, 5 O'CLOCK.

The Secretary moved that the reading of the Minutes of the last meeting be dispensed with, as they had been published in the Transactions. (Carried.)

The report of the Secretary-Treasurer was presented. The Auditing Committee certified to its correctness, and it was accepted and placed on file.

Dr. W. H. Wilder, Chairman of the Theses Committee,

stated that the Committee had received during the year nine theses from candidates for admission to the Society. The Committee had not made any recommendations to the Council, as it was impossible to hold a meeting of the full Committee prior to the meeting of the Council to consider and formulate a plan and ask for amplifications of its powers. Briefly, the Committee desires the Council to empower it to outline a certain definite standard that candidates shall follow in presenting theses. The Committee has not set a standard that requires that the theses offered shall contain the result of original research work, although such a presentation would be welcomed. The Committee feels, however, that a standard high enough should be adopted, that when the theses shall be published subsequently in the Transactions they will be a distinct credit to the Society. The Committee desires to have the power of sending to prospective candidates some definite instructions with regard to the preparation of the theses. Furthermore, it might be well to give the Committee power that, when a man has presented a thesis and it is quite apparent that he could do better, to communicate with him, asking him to amplify the paper and bring it up to the standard required by the Committee.

It was moved that the Society authorize the Council to increase the powers of the Committee on Theses. This motion was carried.

Dr. Myles Standish, Chairman of the Committee of this Society on the Joint Board to Arrange, Control, and Supervise Examinations to Test Preparation for Ophthalmic Practice, presented the following plan of organization as drawn up by the Joint Board, composed of members of the three societies—The American Ophthalmological Society, The Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-laryngology:

PLAN OF ORGANIZATION.

ARTICLE I—NAME.

This body shall be known as the American Board for Ophthalmic Examinations.

ARTICLE II—MEMBERSHIP.

It shall consist of nine members chosen by the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-laryngology; one member to be chosen each year by each of these organizations, in the same manner as its presiding officer, who shall serve for a term of three years. Any vacancy for an unexpired term shall be filled by the Society whose representative has left such vacancy. The terms of members shall begin at the first meeting of the calendar year next succeeding their election; but members elect shall be privileged to attend all meetings of the Board. No member of the Board shall serve more than six years continuously.

ARTICLE III—ORGANIZATION.

At the first meeting of each calendar year the Board shall elect from its membership a Chairman, a Vice-chairman, a Secretary, and three Committees:

(1) A Committee on Finance, consisting of the Chairman and two members.

(2) A Committee on Requirements, consisting of three members, and

(3) A Committee on Examinations, consisting of three members.

The Board shall also designate a financial institution to act as Treasurer, and shall elect an Assistant Secretary who need not be a member of the Board.

ARTICLE IV—DUTIES OF OFFICERS.

The Chairman shall preside at all meetings of the Board and shall prepare the annual report of the Board.

The Vice-chairman shall, in the absence or disability of the Chairman, perform his functions.

The Secretary shall keep correct minutes of the proceedings of the Board, and shall supervise the care of all records of the Board, and the work of the Assistant Secretary, and shall sign all warrants drawn on the Treasurer.

The Assistant Secretary shall be a salaried officer, employed by the Board, who shall conduct its correspondence, receive payments made to the Board, give receipts of the same, and make the proper records thereof; and shall draw all warrants on the Treasurer as authorized by the Board, present them to the Secretary for signature, and shall make and preserve records of the same, and present them upon demand to the Finance Committee; and such other duties as the Board may determine.

The Treasurer shall receive all money paid to the Board and its officers, and shall pay out the same by the order of the Board as evidenced by warrants properly drawn and signed.

The Secretary and Assistant Secretary shall be required to give bond for the faithful performance of their duties, the amount thereof to be fixed by the Board.

ARTICLE V—DUTIES OF COMMITTEES.

The Finance Committee shall each year examine the accounts of all financial officers and agents of the Board, compare the same with vouchers and deposits, and report thereon to the Board; and perform such other services as the Board may direct.

The Committee on Requirements shall (subject to the approval of the Board) formulate the educational and other requirements for those seeking to take examinations in ophthalmology; it shall also be its function to investigate the teaching facilities, curriculum and practice of institutions, and of individual instructors giving instruction in ophthalmology; and shall rate the relative value of such instruction, and prepare records of such ratings for the use of the Board.

The Committee on Examinations shall arrange places, dates and facilities for all examinations held under the authority of the Board; shall frame rules for such examinations (subject to the approval of the Board), shall nominate to the Board examiners for each examination, prepare the

questions for the written examinations, and report to the Board the results of such examinations: such reports to become a part of the records of the Board.

ARTICLE VI—APPLICATIONS FOR EXAMINATIONS.

Every applicant for examination under the Board shall file with the Assistant Secretary a formal blank, prepared for the purpose, giving educational qualifications and professional experience, with a signed photograph of the applicant, and the examination fee. If qualifications are not acceptable to the Committee of Requirements such fee shall be returned.

Any accepted applicant may appear for examination at any regular examination under the Board within five years after such acceptance, by giving sixty days' notice of his intention to do so. Any applicant who takes an examination and fails to pass it shall be entitled, on due notice, to re-examination within three years without additional fee.

ARTICLE VII—RECORDS OF EXAMINATION.

The examiners shall report to the Committee on Examinations their findings in the case of each applicant appearing for examination. These shall be filed with the permanent records of the Board, and shall be accompanied by the answers submitted in written examinations. Such answers shall be preserved for a period of three years and then destroyed. At least once each year the Committee shall prepare for publication a list of all applicants who have passed the examinations since the last preceding list was published.

ARTICLE VIII—CERTIFICATES.

Each applicant who is reported to the Board as having successfully passed the examinations shall receive a certificate to that effect as follows:

The American Board for Ophthalmic Examinations hereby certifies that....., a graduate in Medicine of the....., has pursued an acceptable course of graduate study and clinical work, and has successfully passed the examinations in Ophthalmology conducted under the authority of this Board.

.....19

ARTICLE IX—FUNDS AND EXPENDITURES.

All monies which are received from fees of accepted applicants and from other sources, and which are to be devoted to the purposes of this Board, shall be paid to the Treasurer to be held at the disposal of the Board, as evidenced by warrants signed by the Secretary.

All expenditures of the funds of the Board shall be authorized by formal action of the Board as recorded in its minutes, and shall be for expenses of maintaining an office and preserving records—salary of Assistant Secretary, expenses of place and facilities for examinations and expenses of examiners, and for such other expenses as may be authorized by the Board.

ARTICLE X—MEETINGS.

Stated meetings of the Board shall be held at times and places previously fixed by the Board. Special meetings may be called by the Chairman upon request of a majority of the Board.

A majority of the members shall constitute a quorum for the transaction of business. But no alteration of these by-laws or standing rules shall be effective unless it receive the affirmative vote, either verbal or written, of two-thirds of all members of the Board.

The Chairman of the Board may, by correspondence, upon request of two members, submit any question to a written vote of the members of the Board; and upon favorable ballot of two-thirds of the members may decide it carried.

At stated meetings of the Board, unless otherwise ordered, the following order of business shall be followed:

1. Reading of the minutes of the previous meeting.
2. Report of the Chairman, Secretary and Treasurer.
3. Report of the Committee on Finance.
4. Report of the Committee on Requirements.
5. Report of the Committee on Examinations.
6. Report of the special Committees.
7. Action on Reports of Committees on Examinations with regard to applications for certificate.
8. Unfinished business.
9. New business.
10. Adjournment.

At the conclusion of the reading of the Report Dr. Standish stated that the Board expects that the plan, if successful, will be self-sustaining, and that there will be sufficient funds to meet all expenses. Prior to that time, however, funds will be required for printing and postage, and the committee asks that the Society appropriate the sum of $100 for the use of the Board. Each of the other two constituent societies will be asked to appropriate a like sum to defray the expenses of the Board.

Dr. H. D. Bruns: I would like to ask whether the plans formulated by the Joint Board propose that all those who are now practising ophthalmology shall take the examination; whether those who do not submit to the examination are debarred from registering by the Board as proper practitioners of ophthalmology; and whether a plan had been proposed for a review of any action which the Board might take. He desired to know if a physician, who felt that the action of the Board was unfair, could apply to any of the constituent societies and have a review of his case.

Dr. Standish: No one is obliged to submit himself to the examination; the matter is entirely voluntary. The members of the Board fully discussed the question as to who should be examined, and it seemed to them that the men now established in the profession would not suffer if they did not have the certificate of the Board; consequently they need not take the examination unless they wish to. Of course, if a person did not submit to the examination he would not have the certificate of the Board. As to appeal from the decision of the Board, this question had not yet been considered.

Dr. Lancaster: The Committee on Examinations decides whether a candidate has or has not passed. If he wishes to make an appeal, it is made to the Board, of which the committee is a small part.

Dr. Standish, in reply to a question of Dr. Johnson,

stated that, as the Board is composed of representatives from the three societies, its personnel is changed each year by the new member which each society elects to represent it.

Dr. Jackson: The societies have the power of withdrawing from the agreement at any time, and they have the power of changing the resolution which was adopted that membership in the society shall be conditioned on having passed the examination of the Board. While this Society can at any time withdraw from the agreement, it cannot modify the nature of the Board or its actions unless the other societies are agreed. It is a joint agreement between the three societies, although as a matter of fact all but two members of the Board belong to this Society, all belong to the American Medical Association, and all but one belong to the American Academy of Ophthalmology and Oto-laryngology, and the members of this Society could be instructed to support a definite policy in the Board. It would seem that the only method of appeal would be to the general government of the societies. It will be five years before this movement can be fully on a working basis, and in that time there is opportunity for this Society to consider the plan carefully and decide what course it desires to pursue in regard to it.

Dr. Standish, in answer to a question of Dr. Risley, said that it was the purpose of the Committee of this Society to present a report annually of its recommendations. The proposed requirements have not been given, because the Board desires to take sufficient time to determine what is practical and what is not.

Dr. Wilder: The plan of organization has to be adopted by the other two societies, and until then the committee can only formulate its ideas.

The question of the adoption of the plan of organization as read was put and adopted. The sum of $100 was voted toward defraying the expenses of the Board.

Dr. W. B. Lancaster, Chairman of the Committee on

Standardization of the Illumination of Test Cards and Perimeters, stated that the Committee had held no meetings during the year. The work had really been in the hands of the Chairman of the Joint Committee, who is a member of the Illuminating Engineering Society, and he has made a report, which is printed in the Transactions of that Society. Dr. Lancaster believed that it would be a factor in increasing the efficiency of the Committee if the members were nearer together, and he therefore asked to be relieved from serving on the Committee.

The report of the Council was presented by the Chairman, Dr. W. H. Wilmer:

The Council offered the following nominations of officers of the Society for the ensuing year.

President: Dr. Peter A. Callan, New York.

Vice-President: Dr. Wm. H. Wilder, Chicago.

Secretary-Treasurer: Dr. Wm. M. Sweet, Philadelphia.

It was moved and seconded that the Secretary cast a ballot for the nominees, which motion was carried, and the officers were declared elected.

The Council recommended the following named candidates for admission to the Society, and they were declared elected Associate Members.

Dr. John M. Wheeler, New York.

Dr. Derrick T. Vail, Cincinnati, Ohio.

Dr. Eugene M. Blake, New Haven, Conn.

Dr. Francis W. Shine, New York.

Dr. Clifford B. Walker, Boston, Mass.

Dr. John H. Ohly, Brooklyn, N. Y.

The Council also referred the names of other candidates to the Council for 1917.

The Council nominated the following members of the Society to serve on the Board of Governors of the American College of Surgeons: Drs. Myles Standish, Wm. H. Wilder and Charles W. Kollock.

In compliance with the provisions of the Joint Report of committees of this Society, the section on Ophthalmology of the American Medical Association and the American Academy of Ophthalmology and Oto-laryngology to arrange, control, and supervise examinations to test preparation for ophthalmic practice, the Council recommended for election to the Joint Board Dr. J. E. Weeks, to serve for three years. The Chair announced the election of Dr. Weeks. The representatives of this Society on the Joint Board for the ensuing year are as follows: Dr. J. E. Weeks, to serve for three years; Dr. Myles Standish, to serve for two years; and Dr. Alexander Duane, to serve for one year.

The Council endorsed the recommendation of the Secretary-Treasurer that the present plan of publishing the Transactions in parts, the issues for three years forming the completed volume, be abandoned, and the Transactions for 1917 be published in a separate volume bound in cloth. Upon motion the recommendation of the Council was agreed to. The issue for 1916 will be sent out as usual in paper cover, and form Part II of Volume XIV.

The Council recommended that the satisfactory theses of candidates for membership should be published in the Transactions.

Owing to the increased cost of publication of the Transactions the Council fixed the annual subscription of members for the current year at $10.

The President announced the following Committees:

Council: Drs. R. A. Reeve, Alex. Quackenboss, Arnold Knapp, Wm. Zentmayer, and C. F. Clark.

Committee on Theses: Drs. W. R. Parker, George S. Derby, and W. H. Wilmer.

Program Committee: Drs. W. T. Shoemaker, T. B. Holloway, and the Secretary.

Publication Committee: Drs. W. E. Lambert, Robert L. Randolph, and the Secretary.

Committee on Standardization of the Illumination of Test Cards and Perimeters: Drs. Wm. Zentmayer, Geo. S. Crampton, and H. Maxwell Langdon.

The Secretary announced the death, during the year, of the following members of the Society:

Dr. B. Joy Jeffries, Boston, Mass.

Dr. Edmund C. Rivers, Denver, Col.

Dr. B. L. Millikin, Cleveland, Ohio.

Dr. C. H. Beard, Chicago, Ill.

The Chair appointed Dr. F. E. Cheney, W. E. Bruner, and W. H. Wilder to draft suitable memoirs for publication in the Transactions.

A communication was read inviting members of this Society to attend the fourth annual meeting of the Pacific Coast Oto-Ophthalmological Society to be held in Portland, Oregon, on June 22, 23, and 24, 1916.

A letter was read from Dr. S. C. Ayres, presenting his resignation as a member of this Society. Upon motion it was accepted with regret.

Dr. Edward Jackson proposed the following change in Art. 3, Sec. 1, of the Constitution: After the word "title" in line 7 omit "or shall make public announcement by a sign of a specialty with which he is identified, or shall announce in print that he gives special or exclusive attention to special practice, save only as this affects the Directory of the American Medical Association or similar professional directories"; so that the section shall read as follows:

> The members shall be graduates in medicine, in good professional standing, who have pursued an adequate course of study in Ophthalmic Science and Art. No member shall attach or suffer to be attached to his name, in any public manner, the title of "oculist," or any similar title. The limit of membership shall be 200.

According to the By-Laws the proposed change will come up for consideration at the next meeting of the Society.

MORNING SESSION, MAY 10TH.

The meeting was called to order at 9 o'clock, Dr. de Schweinitz presiding.

Exhibition of Instruments and Apparatus.

18. Dr. A. E. Ewing exhibited "A Practical Lamp for Ophthalmic Operations." Dr. Ewing also showed "A Uniform Visual Test-Object."

Discussed by Dr. Holt.

19. Dr. George S. Crampton demonstrated a case of "An Unusual Type of Retinal Pigmentation."

Discussed by Drs. Zentmayer, Weeks, Lancaster, and Crampton.

20. "Tuberculosis of the Retinal Vessels." Edward Jackson, M.D.

Discussed by Drs. Verhoeff, Weidler, Fisher, Knapp and Jackson.

21. "A Clinical and Pathological Study of Two Cases of Miliary Tubercles of the Choroid." Robert L. Randolph, M.D., and Harry C. Schmeisser, M.D. (by invitation).

Discussed by Drs. Risley and Randolph.

22. "Reports of Cases of Quinin Amblyopia with Remarks." John E. Weeks, M.D.

Discussed by Drs. Bruns, Ellett, Fisher, de Schweinitz and Weeks.

23. "Retrobulbar Neuritis with Central Scotoma from Toxic Action of Thyroidin." Myles Standish, M.D.

24. "Optic Neuritis from Syphilitic Leptomeningitis." William H. Wilder, M.D.

25. "Cyanosis Retinæ et Conjunctivæ in Connection with Pulmonary Stenosis and Patent Ductus Arteriosus." Robert Scott Lamb, M.D.

Discussed by Drs. Holloway, Weidler, and Lamb.

26. "A Case of Pituitary Body Disease with Reference

to the Effects of Early Operation." Wm. Evans Bruner, M.D.

Discussed by Drs. de Schweinitz and Bruner.

27. "High Hyperopia from a Clinician's Standpoint." Howard F. Hansell, M.D.

28. "Unusual Complications in a Case of Glaucoma after Atropin." T. Y. Sutphen, M.D.

29. "A Case of Botulism." Walter B. Lancaster, M.D.

30. "Isolated Paralysis of the Cervical Sympathetic Accompanying Goiter, with Report of Two Cases." H. Maxwell Langdon, M.D.

31. "Double Papillitis, Presumably of Tonsillar Origin." Andrew Timberman, M.D. (Read by title.)

Adjourned.

The following members were present and registered at the Fifty-second Annual Meeting:

Alger, Ellice M.
Alling, Arthur N.
Bailey, Fred. D.
Bedell, A. J.
Bell, James H.
Bordley, James, Jr.
Brown, E. V. L.
Bruner, Wm. Evans
Bruns, H. Dickson
Butler, W. K.
Callan, Peter A.
Carmalt, W. H.
Carpenter, John T.
Carroll, James J.
Chance, Burton
Crampton, George S.
Curdy, R. J.
Derby, George S.
Dodd, Oscar
Duncan, J. L.
Ellett, E. C.
Ewing, A. E.
Fisher, Carl
Fiske, George F.
Foster, Matthias L.
Fox, William H.
Fridenberg, Percy
Goldberg, Harold G.
Greene, Louis S.
Greenwood, Allen
Hansell, Howard F.
Harlan, Herbert
Harrower, David
Heckel, Edward B.
Holloway, T. B.
Holmes, C. R.
Holt, E. E.
Hunter, D. W.
Jack, Edwin E.
Jackson, Edward

Jameson, P. Chalmers
Johnson, W. B.
Kennon, B. R.
Kilburn, Henry W.
Knapp, Arnold
Kollock, Charles W.
Lamb, Robert Scott
Lambert, Walter Eyre
Lancaster, Walter B.
Langdon, H. Maxwell
Loring, Francis B.
Loring, Robert G.
Lovell, D. B.
Marlow, F. W.
Marple, W. B.
Maxson, S. C.
May, Charles H.
Miles, Henry S.
Parker, Walter R.
Posey, W. C.
Price, Henry R.
Pusey, Brown
Pyle, Walter L.
Quackenboss, Alex.
Radcliffe, McCluney
Randolph, R. L.
Ray, J. M.
Reeve, Richard A.
Reik, Henry O.
Reynolds, W. G.
Ring, G. Oram
Ring, Henry W.
Rogers, W. K.
Roy, Dunbar
Risley, J. Norman
Risley, Samuel D.
Schneideman, T. B.
de Schweinitz, G. E.
Shannon, C. E. G.
Sherman, H. G.
Shoemaker, J. F.
Shumway, Edward A.
Smith, E. Terry
Snell, Albert C.
Spalding, F. M.
Spratt, Charles N.
Standish, Myles
Sutphen, T. Y.
Sweet, Wm. M.
Taylor, Lewis H.
Theobald, Samuel
Thorington, James
Török, E.
Timberman, Andrew
Veasey, C. A.
Verhoeff, F. H.
Waite, Frank L.
Weeks, John E.
Weidler, Walter Baer
Wescott, Cassius D.
White, J. A.
Wilder, Wm. H.
Williams, Edward R.
Wilmer, W. H.
Woods, Hiram
Zentmayer, W.
Ziegler, S. Lewis

THREE YEARS' EXPERIENCE IN SCLEROCORNEAL TREPHINING IN GLAUCOMA.

W. H. WILMER, M.D.,
Washington, D. C.

The presentation of my experience with sclerocorneal trephining based upon so limited a number of cases must find its excuse in the fact that, by very reason of the small number, I have been able to personally keep in touch with their after-histories. Thus it has been possible to carefully watch these cases, almost without exception, at intervals after the operation, to have the tension taken, the central vision recorded, and the fields charted. Some of these patients were operated upon before Col. Elliot's visit to this country, but the majority were operated upon shortly afterward, owing to the interest aroused by the press accounts of his work. His operation gave a ray of hope to a number of people who had been gradually losing ground in spite of miotics, and in some cases in spite of previous operations. I will not take the time of the Society to go minutely into any of the individual cases except where there has been a return of tension or some other complication.

Of the three cases of chronic glaucoma with acute exacerbations, my first patient was a rather frail woman, fifty-six years of age, who had already lost one eye, the right one. It was in a state of absolute glaucoma, with a tension of 58 mm. The tension in that eye was permanently relieved by a Lagrange operation with iridectomy. The patient suffered from an acute attack of glaucoma in the left eye, during which time the vision was reduced to counting fingers. After the acute attack the tension did not fall below 35 mm. Two Lagrange operations at six weeks' intervals, the first

being associated with an iridectomy, relieved the tension only temporarily. Six weeks after the last Lagrange a trephine operation without iridectomy was done to the nasal side of the site of the former operations. There was a slight opacity upon the upper anterior part of the lens-capsule following the Lagrange operation and previous to the trephine. This has not increased. The central vision is 20/30−, the field normal, and the tension 8 mm. There is a large bleb over the trephine site.

The second case of this type occurred in a woman sixty-eight years of age with immature cataract. The tension after recovery from the acute attack was 29 mm. The operation had no noticeable effect upon the cataract. The tension was reduced to 14 mm.

The third case of this type occurred in a woman, aged seventy-five years, whose right eye had been removed. Her physician stated that the eye had been lost from glaucoma, and enucleation had been resorted to in order to relieve great pain. The sight of the left eye was merely light perception. She had suffered from a number of acute attacks of glaucoma. The tension between attacks was 52 mm. She desired the trephine in lieu of enucleation to relieve pain. There has been no pain since the operation, the tension is 6 mm., the sight unchanged.

Mr. N., aged sixty-one years, has suffered from chronic inflammatory glaucoma for many years, during which period he faithfully used a miotic. V.R.E. = 2/200; L.E. = 6/200. Fields much contracted. In trephining, a peripheral iridectomy was performed on the right eye and a complete iridectomy on the left. The patient recovered normally from a severe attack of acute contagious conjunctivitis in both eyes several months after the operation. The central vision improved, the fields enlarged, and the tension was 10 mm. in both eyes.

In the group of cases of secondary glaucoma following iridocyclitis my first patient was a man, twenty-eight years of age, who developed glaucoma after a long-standing iridocyclitis from a blow on the eye. The lowest tension recorded during his glaucomatous period was 35 mm. He suffered greatly from pain. The pain was quickly relieved by the operation, and the tension remained 10 mm.

The second patient came to me with the history of a long-standing iridocyclitis after cataract extraction. She had been suffering great pain for many months. The right eye had light perception, the left eye was blind. The tension of the right eye was 35 mm., that of the left eye 85 mm. Trephining was done on both eyes under a general anesthetic. After twenty-four hours the pain disappeared and has not returned. Now, two and one-half years after the operation, the tension of the right eye is 8 mm., that of the left, 13 mm. The vision is unchanged, except the light perception is a trifle better in the right eye.

The third case of this series occurred in a man aged sixty-one years. There was mere light perception, and the operation was done to relieve pain and in place of enucleation. The pain has been entirely relieved and the tension reduced to 7 mm.

The fourth patient of this type was a man, sixty-six years of age. One eye was affected. The pain was relieved and the tension reduced from 38 mm. to 20 mm. by the operation.

The last case of this series, a woman of sixty-six years, came to me with the history of a post-operative iridocyclitis. The tension just before the trephining was 55 mm. When last taken, it was 9 mm. However, this case was trephined on too recent a date to be sure that the success is permanent.

I have done this operation on only one case of glaucoma secondary to retinal hemorrhages. This patient, a man aged fifty-eight years, suffered from absolute glaucoma of the right eye with repeated corneal ulcerations. His blood-pressure was high and the intraocular tension was 70 mm. The eye was continually irritable and often very painful, yet he wished to avoid enucleation. The trephining was done in the hope of avoiding the removal of the eye. The anterior chamber was abolished, the tissues very friable, and it was impossible to tap the anterior chamber as in the more normal cases. A complete iridectomy was also done. There was a trifling loss of vitreous, but the healing was smooth and the pain quickly relieved. However, two and one-half years after the trephine there was a depression over the site of the wound and the globe was shrunken. The eye was removed with fat implantation in Tenon's capsule.

In one case of glaucoma following anterior adherent

leukoma, with partial staphyloma of the cornea and lead deposit on the scar, I did a trephine with a later removal of the metallic deposit and a tattooing of the scar. This was done for a relief from discomfort, and for cosmetic reasons in lieu of enucleation. The discomfort from the bulging, irritated cornea is entirely relieved and the eye is no longer disfiguring. The tension has been reduced from 34 mm. to 8 mm. Patient exhibited.

I have performed sclerocorneal trephining in only one case of glaucoma occurring after cataract extraction where no other complication existed. This is not a typical case of glaucoma due to the extraction, because it also ensued in the other eye which had not been operated upon. It does, however, illustrate the ease with which this operation can be performed in similar cases.

In May, 1903, I extracted a cataract from the right eye of Mr. B., aged sixty-five years. A broad iridectomy was done at the time. Operation and recovery smooth. No incarceration of capsule or iris pillars in the wound. Vision 20 40+. In June, 1915, twelve years later, the patient returned with the report that the vision in the right eye had been failing for the last year or so. V.R.E. = 20 70−, fields contracted, tension 38 mm. The trephining was done over the site of the former corneal section. The fields and the vision remained the same after the operation, but the tension was reduced to 10 mm.

In the group of chronic simple glaucoma, one case had a temporary return of tension in one eye. This patient, a woman aged forty-six years, had normal central vision, but the fields were small and contracted almost to the fixation point. Tension, R.E., 42 mm.; L.E., 34 mm. The central vision remained normal, but the tension continued high and the fields lost in spite of active treatment with miotics. A sclerocorneal trephining was done in both eyes with a peripheral iridectomy. The operation and recovery were smooth. The central vision continued normal, the fields enlarged somewhat, and the tension continued low. On December 11, 1915, over a year after the operation, the patient's eyes were examined in the morning. Tension, R.E., 11 mm.; L.E., 6 mm. No miotic had been used for over a month. That same afternoon the patient sewed

with feverish haste for several hours, continuing into the twilight. That night she had an acute attack of glaucoma in the right eye. By the use of miotics the pain soon ceased, but two days later the tension was still 36 mm. On January 3d, twenty-three days after the attack, the tension was reduced to 20 mm. and is at present 19 mm. During this time the tension of the left eye remained about 6 mm. The patient is doing incessant and close eye work, but the eyes continue in good condition. She is using a mild miotic in the right eye twice a day.

The second patient of this group unfortunately furnishes two eyes as examples of return of tension, after the lapse of some time, following a seemingly perfect sclerocorneal trephining. She is forty-eight years old, thin and frail looking, but with much courage. Her father and one brother died from tuberculosis. Her mother died of throat trouble. One aunt had a tumor. The patient had suffered from pyorrhea, had never had any illness until the grip five years ago. Occasionally anemic, mitral stenosis with good heart functioning. Some auricular fibrillation. Old extensive and, probably, latent or inactive pulmonary tuberculosis. The kidney elimination of phenolphthalein is low—36 per cent. in two hours. Slight trace of albumin. Hemoglobin, 85 per cent. Red blood-cells, 3,816,000; white, 10,000. Wassermann negative. Blood-pressure, 135–85. She was first seen on February 18, 1913. She stated that the left eye had been failing for a number of years. Anterior chambers shallow, pupils moderately dilated. Marked cupping of both discs, atrophy being pronounced in the left eye. Field of the right eye a trifle contracted, that of the left eye markedly so, superiorly and nasally almost to the point of fixation. V.R.E. = 20/20; L.E. = 20 50−. Tension, R.E., 31 mm.; L.E., 42 mm. Miotics were used strenuously for over four months. The central vision remained the same, but the fields contracted. On June 24, 1913, the left eye was trephined, with a small peripheral iridectomy. Operation and recovery were smooth. The central vision of that eye improved to 20/20−, and the fields enlarged. The average tension was 15 mm. Col. Elliot saw this eye when he was in Washington, and used the patient to illustrate a case of perfect drainage after his operation. He also strongly ad-

vised a trephine upon the other eye. On December 20, 1913, this was done with a small peripheral iridectomy. The operation and recovery were smooth. In April, 1914, the patient had an acute contagious conjunctivitis in both eyes. She recovered without any complication. In May, 1914, V. R. E. = 20 20; L. E. = 20 20 −. Fields enlarged; tension in each eye 13 mm. On December 21, 1914, a year after the operation on the right eye, the tension of that eye rose to 36 mm., the tension of the left remaining 17 mm. After using $\frac{1}{2}$ per cent. solution of pilocarpin and hot applications every two hours for two days the tension fell to 16 mm. It then gradually rose again, varying from 36 mm. to 61 mm. During this period the left eye, the one first operated upon, remained below 20 mm. On January 19, 1915, a second trephine was done on the right eye, to the temporal side of the previous opening; a small peripheral iridectomy was also done. Operation and recovery were smooth. The sight and tension remained satisfactory. The fields showed no further contraction. On December 8, 1915, nearly a year after the second trephine on the right eye, the tension of that eye again increased to 34 mm., that of the left eye remaining normal. On March 17, 1916, V. R. E. = 20 20 −; L. E. = 20 30 −. Tension, R. E., 43 mm.; L. E., 36 mm. It will be seen that the left eye after perfect quiescence and drainage for three years, lacking three months, again ceased to drain properly. At this time the eyes presented macroscopic anatomical changes. The anterior chamber became almost abolished in the right eye, and more shallow in the left, the translucent blebs, with uplifted conjunctival and subconjunctival tissue, partially disappeared, and the trephine opening seemed to be occupied by a pigmentary deposit. At the same time the site of the trephine openings, which originally seemed perfectly placed, gave the impression of having been located too far back, leaving a space of scleral tissue between them and the corneal margin; whereas, in fact, in each case the drill overlapped well into the corneal tissue. The fields, which had increased during the period of freedom from high tension, began to contract. A second trephine on the left eye was determined upon and a third upon the right eye, with a complete iridectomy through the trephine opening. This was done on April 18, 1916. In

the left eye this was accomplished without difficulty, the aqueous immediately escaping after the trephine pierced the sclera. The iris ballooned into the wound and was easily excised. In the right eye the operation was more difficult owing to the obliteration of the anterior chamber. The aqueous did not escape until the iridectomy was done. So far as the eyes are concerned, the recovery has been painless and smooth. However, on the ninth day after the operation a cerebral hemorrhage occurred, causing a left hemiplegia. The patient, however, is improving, and the eyes are soft and comfortable. I had hoped to be able to show this patient to the Society.

In the case of Mr. N., aged fifty-three years, we have an illustration of the successful cases of this group. High degree of hyperopia; glaucoma simplex both eyes; V.R.E.= 20 20; L.E.=20 200. Tension, R.E., 33 mm.; L.E., 57 mm. Fields markedly contracted nasally and above in the left eye. December 3, 1912, Lagrange with iridectomy performed on the left eye. For a year the tension with the use of miotics remained about 28 mm. It then gradually increased to 50 mm., the tension of the right eye going up to 43 mm. The patient had continuously high blood-pressure, at times as much as 280. On December 9, 1913, both eyes were trephined and an iridectomy performed on the right eye. When the trephine was completed on the left eye, the aqueous spurted through the top of the drill. There was much chemosis of the ocular conjunctiva of both eyes for five or six days after the operation. Mild iritis in the right eye, none in the left eye. V.R.E.=20 20; L.E.=20 40−. Fields improved. Tension, R.E., 16 mm.; L.E., 14 mm. Patient exhibited.

Of this series, one patient had a rupture of the conjunctiva over the trephine opening eight months after the operation. This was repaired by a sliding flap of conjunctiva. One patient had marked vitreous opacities and incipient cataract. There has been no increase of the cataract since the operation two and one-half years ago.

Of the twenty-seven eyes with glaucoma simplex, there has been a serious return of tension in two eyes (in the same

person). I hope that this percentage in glaucoma simplex (7.4 per cent.) may be eventually reduced by the last operation.

The good results in the successful cases cannot be attributed to the iridectomy alone; for among my cases five had undergone an iridectomy, and four of them a Lagrange with iridectomy, previous to the trephining. In all of these nine eyes there had been only a temporary relief of tension, though a little drainage seemed to be still going on. Since the trephining there has been no return of tension in any of these nine eyes.

In all of these cases, before resorting to operation, miotics were strenuously used for a period of time covering years, in some cases.

The advent of the tonometer marks an era in the history of glaucoma; but I believe firmly that very careful palpation should be practised even more than ever in order to verify the findings of the tonometer; for some of the newer instruments, in my own experience, have not proved themselves correct. My original Schiötz instrument is more reliable than any of the later models, and its measurements tally more closely with palpation and the clinical phenomena of glaucoma. It seems trivial to speak of the method of using the familiar tonometer, but the carefulness of that method cannot be too strongly emphasized. The patient should lie practically flat on the back and relax; holocain should be instilled several times; sufficient time should be allowed for complete local anesthesia. When possible, an assistant should hold the lids in order that the orbicularis may not alter the results by pressure on the globe. At least three weights should be used, and the instrument manipulated with such deliberation that the surgeon may be able to take the mean of the systolic and diastolic intraocular tension as shown by the slight variation of the indicator.

Shortly after the introduction of this operation it was written about and discussed with much enthusiasm. Now

it seems as though the pendulum of professional opinion had a tendency to swing a bit too far in the other direction. The operation is objected to on the ground that synechiæ are apt to follow it, but that is also true of other operations used for the relief of tension, such as iridectomy and the Lagrange. A complete iridectomy at the time of the trephining, with the proper use of a mydriatic, will reduce this tendency. This operation has been condemned also because lenticular opacities have followed its performance. Cataracts in older people are apt to be associated with glaucoma, or glaucoma with cataracts. I have not seen any cataracts caused by the operation, nor do preëxisting cataracts seem to have increased with unusual rapidity afterward. It is possible to cause a cataract by the drill, but it is possible to cause it in other operations that are not skilfully performed.

Late infection has occurred with unpleasant frequency, according to most reliable reports. I believe that this danger can be lessened by taking great care to dissect up all of the subconjunctival tissue with the conjunctiva, in order to make the flap as thick and protective as possible. I warn my patients of the possibility of infection, and advise a boric acid solution to be used twice a day even when no miotic is used. I also give them directions (with necessary prescriptions for argyrol, etc.) for the care of the eyes in case of an acute conjunctival inflammation until expert advice can be obtained. Of course, prophylaxis is more difficult among ordinary hospital patients.

Hemorrhages into the vitreous from the choroid or the retina have occurred in the hands of the most skilful operators. This danger can be lessened by using certain precautions before the operation, such as decreasing excessive blood-pressure; reducing the intraocular tension by the strenuous use of miotics; avoiding a general anesthetic and giving morphin hypodermically in suitable cases; the

administration of lime salts in cases of delayed coagulation. However, intraocular hemorrhage is a danger in all procedures where the tension of the eye is suddenly reduced.

Relapses, too, do occur, as I know to my discomforture; but no procedure, surgical or non-surgical, is exempt from that calamity.

Weighed in the balance of my own personal experience, the operation of sclerocorneal trephining presents many advantages. Both eyes can be operated upon at the same time and the patient does not have to be very quiet or very long confined. The operation can be done with ease, except in cases where the anterior chamber is abolished. It does not reduce the visual fields, therefore it can be done in those cases where the restriction is almost to the visual center. It has not immediately reduced vision in any case, but it has improved the sight in a number of cases. It has not caused astigmatism to any accountable extent, and therefore it can be safely done where the vision is normal without any undue risk of immediate impairment of sight. There is only a negligible risk of the loss of vitreous and no danger of the escape of the lens. The operation may be repeated.

There is no ideal operative procedure, but in my limited experience, for permanently reducing excessive tension in all cases of glaucoma, except the acute form, sclerocorneal trephining is the easiest, safest, and most effective method so far presented to ophthalmic surgery.

DISCUSSION.

DR. W. B. MARPLE, New York: An experience with the operation of three years has led me to the same conclusions as those of Dr. Wilmer. There is only one point to which I would like to refer, and that is to its relative safety in cases of chronic glaucoma with a very narrow nasal field. If an iridectomy is done on these cases, we cannot be at all sure as to what will be the outcome. I would like to show a field in a case of chronic glaucoma in which a large atrophic

element was present. I saw the patient in May, 1914, and at that time the field was considerably larger, and I proposed trephining. Nothing was done until February, 1915, when this field was taken and the vision was 20 70. A colleague then did a most skilful Lagrange operation, with a broad iridectomy, and its effect in the way of reduction of tension was most satisfactory, but the field, previously very narrow, became further contracted, so that before the patient left the hospital she had lost the fixing-point and vision was reduced to fingers at five feet, eccentric. I cannot but feel that had a trephine operation with a peripheral iridectomy been done upon this patient, her vision would have been preserved. If a broad iridectomy is done in a case of chronic glaucoma with narrow nasal field, the risk of the loss of the rest of the nasal field is very great.

I also show the fields of two patients with narrow nasal fields. The one was a man, aged sixty-five years, with corrected vision of 20 40, operated on February 20th. The other was of a lady with corrected vision of 20 70 and operated on March 14, 1916. A trephine operation with peripheral iridectomy was done in each case. The fields after the operation were even larger than before the operation. I have two other operative cases with equally narrow nasal field, where this was a little larger after trephining; one of them was operated on two years ago.

In a general way I agree with Meller that the lowering of the tension in the trephine operation is apt to be greater after a complete than after a peripheral iridectomy. I have in several patients done a complete iridectomy in one eye and a peripheral iridectomy in the other, and have always found the tension lower in the eye with the complete iridectomy. But whenever I have a case in which the nasal field is narrow, I always try to make the iridectomy a peripheral one. I feel confident that had the first patient referred to above had a trephine operation with peripheral iridectomy, she would not have lost the fixing-point. In one or two cases of absolute glaucoma I have made at the same time two trephine openings, about 8 mm. apart, with apparent beneficial effect in the reduction of the tension. I have never had a case of secondary infection following the trephine operation, so that I agree entirely with the con-

clusions of Dr. Wilmer and consider sclerocorneal trephining a valuable addition to ophthalmic surgery.

Dr. H. D. Bruns, New Orleans, La.: My experience, although limited, has been exactly the same as that of Dr. Marple; namely, that where we have a narrow field, the field does not change after the trephine operation. I have one case that I saw just before I left home, in which the vision has stayed within five degrees of the fixation point for two and one-half years after trephining.

Another point I wish to speak of is this: When the operation was proposed, I restricted myself almost entirely to useless eyes, because I wanted to see if I could convince myself as to the relative merits of iridectomy and of sclerectomy in reducing tension; and I have convinced myself that the tension is as much lowered by the sclerectomy without iridectomy as it is with iridectomy. I did all my earlier operations without an iridectomy and found the tension as greatly reduced as though I had employed it. Iridectomy is necessary to prevent circular synechiæ in almost all cases, and to prevent the iris from prolapsing into the wound. A radial snip through the prolapsed iris will cause it to retract within the anterior chamber; the resulting linear scar becomes invisible in the course of a short time and the eye presents no appearance of having been operated upon. An interesting case demonstrating the efficiency of sclerectomy and the uselessness of iridectomy to control tension was that of an old woman whose right eye I trephined for acute glaucoma, *without an iridectomy.* The tension was entirely *relieved* and the eye restored to usefulness. In the left eye there was an immature cataract and a correct preliminary iridectomy, but the incision in the limbus did not prevent increase of tension, which had to be controlled with miotics. I am convinced by my experience that the iridectomy has nothing to do with the reduction of tension, but it is necessary to prevent prolapse of the iris into the trephine hole.

Dr. W. E. Lambert, New York: In connection with this operation I wish to report two cases. The first was one of late infection, occurring in one eye. The patient was a young man who had hereditary glaucoma, his father having been operated upon by Dr. Gruening twenty years previously.

DR. ANDREW TIMBERMAN, Columbus, Ohio: Just a suggestion in regard to the technic of the operation for trephining which I have found of help. Prior to the operation we use a sub-conjunctival injection of the solution of adrenalin for control of the hemorrhage, which we occasionally find is quite bothersome. This idea occurred to me and I have tried it, and find that if we limit the injection to the space directly under the conjunctiva, and not in the sub-conjunctival tissue, it is quite a help and aid to us in the technic of the operation.

DR. G. E. DE SCHWEINITZ, Philadelphia: I am in exact accord with the opinion expressed by Dr. Marple; namely, that corneoscleral trephining is a safer operation than iridectomy when the visual field is much contracted and the blind area encroaches on the fixation point. In a paper presented to this Society two years ago I stated that in chronic glaucoma with greatly contracted visual field it appeared to me that corneoscleral trephining was a safer procedure than ordinary iridectomy, which so often has been followed in these circumstances by a rapid obliteration of this much contracted field. Whether because iridectomy helps in the reduction of the increased intraocular tension or prevents quiet iritis it certainly, as Dr. Marple properly insists, is an important part of the technic of the operation of corneoscleral trephining.

THE OPERATIVE TREATMENT OF PARTIAL STAPHYLOMA OF THE CORNEA AND OF FISTULA OF THE CORNEA WITH A CONJUNCTIVAL FLAP; CONJUNCTIVO-KERATOPLASTY (KUHNT).

ARNOLD KNAPP, M.D.,
New York City.

CASE I.—*Partial Corneal Staphyloma; Recurring Attacks of Glaucoma and Iritis; Excision of Staphyloma; Conjunctivo-keratoplasty. Result: The New Scar Subsequently Became Slightly Ectatic and Boggy and the Increased Tension Returned.*—C. H., aged fourteen years, after a perforating corneal ulcer in the left eye in November, 1911, developed a

Both eyes had been operated upon, one two years ago and the other about eighteen months ago, both operations were uncomplicated and the result most satisfactory. About a week ago I was asked to see this patient at the New York Eye and Ear Infirmary, where he had been taken as a semi-private patient. He had a serious infection of the first eye operated upon. I understand he is progressing satisfactorily, with a good chance of saving the eye and probably some vision.

The second case was one in which this operation was made for an acute attack of glaucoma. The patient was a physician who had lost one eye. He had had several attacks of glaucoma, not particularly acute, and he had been able to control these attacks with miotics. I advised an iridectomy, as I always prefer an iridectomy to a sclerocorneal trephining in acute or sub-acute cases. He recovered from that particular attack and preferred to wait. Some time afterward he had a rather severe attack, and vision was reduced to 20/100, tension, 50 mm. He consented to an operation but insisted upon a sclerocorneal trephining, although I advised strongly an iridectomy. The result was most satisfactory. His vision was restored to 20/20, and he has not had an attack of glaucoma since, which was over a year ago.

DR. A. E. EWING, St. Louis: In operating on three eyes for the relief of glaucoma by the Elliot trephining method, one because of a secondary glaucoma from which the fellow eye had been lost, and the other two because of secondary glaucoma with total staphyloma, the trephine openings were placed to the nasal side above the horizontal meridian about on a line with the upper border of the insertion of the tendon of the internal rectus muscle, this location having been chosen with the purpose of also obtaining vision by the accompanying iridectomy. Relief from the glaucoma resulted in all the cases, with disappearance of the staphyloma in the last two eyes and a great improvement in the vision of the first one, but there was no formation of the characteristic conjunctival bleb. Although there was no true glaucoma in any of these eyes, the lack of the bleb indicates that the neutral region between the insertion of the muscles may be a more proper location for securing drainage than that in line with the tendon.

circumscribed ectatic scar measuring 4.5 mm. vertically and 5.5 mm. horizontally, below and nasal from the corneal center. At one point in the scar there was a translucent area where iris tissue presented and rupture occurred from time to time with each attack of glaucoma. The iris was adherent except up and out, where an iridectomy had been done. V. = 6/200.

An operation was decided upon to safeguard the eye from these attacks of glaucoma and from the inflammation which followed the perforation, and was performed on October 13, 1914, under ether narcosis. A conjunctival flap was prepared from the lower sclerocorneal margin. The ectatic corneal scar was cut away flush with the corneal surface and the iris was freed with a spatula. The conjunctival flap was drawn in place and anchored by sutures at each extremity.

The subsequent healing was uneventful; a tight bandage was constantly applied, the flap healed firmly in place, the anterior chamber was soon restored, and there was no cataract. The tension remained down, except on a few occasions, when eserin had to be given.

On November 11th and 14th the extremities of the conjunctival flap were divided. On November 23d tension was distinctly increased; it looked as if section of the pedicles had shut off a way of filtration for the aqueous. The conjunctiva over the corneal scar was edematous and boggy; at its upper margin there were a number of small transparent vesicles. To reduce the tension a cyclodialysis was done at the upper and outer quadrant at the site of the old iridectomy. This reduced the tension for several weeks and the scar became flat and the vesicles disappeared. On December 12th the scar was again bulging, and there were some deep vessels running to it from the periphery of the cornea. The increased tension has been readily kept down by massage. February 20, 1915: The scar was flatter, though the lower margin overhung the neighboring cornea. It was distinctly edematous and could be indented with a probe. The tension seemed normal. April 27th: The condition had not changed, though possibly the area of the scar was somewhat more prominent. Tn. The lower half of the ectatic scar was diminished by the excision of a superficial crescentic piece of tissue. August 31st: The scar was somewhat

smaller, though it still bulged like a cyst. December 13th: The scar was boggy, the vessels were distinctly to be seen running into the scar from the lower corneal margin; the tension was raised. March 25, 1916: The ectatic scar measured 5.5 x 5, and was elevated 1.5 mm. T.+1. The scar was edematous and it could be pitted. An anterior capsular cataract had developed. The eye was free from irritation and caused no discomfort. The main objection was the unsightliness of the circumscribed opaque protrusion on the cornea.

CASE II.—*Partial Staphyloma of the Cornea; Repeated Perforation after Attacks of Glaucoma; Excision of Staphylomatous Part; Iridectomy; Release of Adherent Iris; Conjunctivo-keratoplasty. Result: A Perfectly Flat and Firm Scar with no Return of Increased Tension or Irritation.*—L. G., aged fourteen years, seven years ago suffered from a corneal ulcer. During the past three years had three attacks of acute glaucoma. Three days ago another attack. At the time of the first visit to the Herman Knapp Memorial Eye Hospital, on June 20, 1915, an adherent leukoma occupied a lower quadrant of the cornea. This part of the cornea was replaced by a thin and ectatic scar with several translucent spots. T.+2. S.=H. M.

Operation July 2d, under ether narcosis. A flap of conjunctiva was detached along the outer margin of the cornea. The staphylomatous area was excised. The attempt to free the iris on each side and to divide it horizontally with a scissors was only partly successful. An iridectomy was done above. The flap was adjusted and held in place by sutures.

July 16th, no reaction; the flap was in place and had attached itself. It was divided above. September 22d, the cornea was flat; the area of the old staphyloma was covered with a flat conjunctival scar. There was no cataract. Tn. The eye was white and had been perfectly comfortable. February 11, 1916, cornea flat. Tn. S.=H. M.

CASE III.—*Corneal Fistula; Adherent Leukoma; Recurring Attacks of Glaucoma; Iridectomy; Curettage of the Fistulous Tract; Conjunctivo-keratoplasty. Result: Fistula Healed;*

Tension Normal.—J. L., aged thirty years; twelve years ago suffered from an ulcer of the left cornea. Has had many attacks of increased tension which were relieved by perforation. V. = 1/200. There was a deep glaucomatous cup. In the outer half of the left cornea there was a small, round, slightly bulging scar with a central dark spot consisting of iris tissue.

Operation March 16, 1915, under local anesthesia. An iridectomy was done at the temporal margin of the cornea, so as to include the iris, which was adherent to the cornea at the site of the corneal fistula. A broad spatula was then introduced into the anterior chamber, and with this as a background the fistulous tract in the cornea was curetted right down to its base. A conjunctival flap was taken from the outer margin and drawn over to cover the fistula, where it was firmly pressed into the fistulous opening and held in place by sutures at the sclerocorneal margin.

March 25th the flap had adhered to the corneal ulcer and was divided below. Two days later this was repeated above. The fistula was perfectly closed and the tension was normal. November 26, 1915, the cornea was firmly healed; tension, 16 mm.; the eye was perfectly comfortable.

Partial staphyloma of the cornea, when complicated with glaucoma and attacks of iridocyclitis, is one of the most difficult problems which confront the ophthalmic surgeon. Either sight is lost from glaucoma, or the patient is subjected to repeated and painful attacks of congestive glaucoma which are relieved by perforation and escape of aqueous, when the danger of a secondary infection and purulent iridocyclitis is present. The only method of treatment which offers some prospect of success consists in excision of the staphylomatous portion of the cornea, in release of the adherent iris with iridectomy, and in covering the defect with a double pedunculated conjunctival flap, as particularly recommended by Kuhnt. The excision of the staphyloma and the conjunctivo-keratoplasty are relatively simple procedures, but the restoration of the normal intraocular tension is the diffi-

cult step and the one upon which the success of the keratoplasty depends. The difficulty of curing a glaucoma secondary to an anterior adhesion of the iris in conditions of adherent leukoma is well known. The important factor in the relief of glaucoma in these cases, to my mind, is the time at which the iridectomy is performed—taking for granted that the iridectomy is complete and peripheric,—as in old and confirmed cases operations are generally not successful. If the tension is not reduced, the result of the keratoplasty may be like the condition described in the first one of my cases. The new scar, though thick, bulges and becomes edematous and boggy as if the aqueous was finding a means of escape through it. Kuhnt* has observed this in a number of cases of conjunctivo-keratoplasty, and regards it as due to the fact that the corneal surface of the conjunctival flap did not heal promptly and that the aqueous constantly penetrated into it. He has, therefore, modified his procedure in these cases by not excising the entire part of the ectatic corneal scar, but removing only the superficial layers and making a linear cut in the deeper layers with a knife. This thin layer of tissue, he believes, is sufficient to keep the aqueous from penetrating the flap. In a later article† Kuhnt says that his original method, consisting in excision of the staphylomatous part of the cornea, conjunctivo-keratoplasty, and iridectomy, is applicable only for early cases. For older and extensive cases he resects a crescent from the periphery of the ectatic corneal scar after removing the anterior layers, sutures this to the adjoining corneal or scleral margin with catgut, and covers it with a conjunctival flap.

I believe that in my first case the increased tension was not corrected because the iridectomy had not been done early enough, as it was performed about one year after the perforating ulcer of the cornea, and that was two years

* Zeitschr. f. Augenheilk., xxiv, p. 19. † *Ibid.*, p. 517.

before the patient came into my hands. At the time of the keratoplasty I neglected to do a tension-reducing operation, and did not obtain a satisfactory corneal scar. I shall make another attempt to reduce the glaucoma with a scleral resection operation and to reduce the size of the thickened flap with partial excision or with the galvanocautery. In my second case a thick, flat scar replaces the previously thin, ectatic one, and the tension of the eye is normal; this satisfactory result was obtained principally because the iridectomy relieved the glaucomatous condition. I am firmly convinced that generally the iridectomy is not done early enough in cases of perforating ulcers of the cornea, which are likely to be followed by glaucoma from the extent of the anterior iritic adhesions. I believe that the iridectomy should be done just as soon as the inflammatory conditions in the cornea permit, and that it is a mistake to wait until the intraocular tension is increased.

The relief of a fistula of the cornea is a simpler condition, and one more amenable to operative treatment. The procedure which Kuhnt recommends,* and which was followed out in the above case, was simple and gave a perfect result. Kuhnt (*loc. cit.*) has reported on 19 cases of corneal fistulæ which he has operated upon with conjunctivo-keratoplasty; of these, 16 were cured, in 3 a second operation was successful, made necessary by a displacement of the conjunctival flap.

DISCUSSION.

DR. DUNBAR ROY, Atlanta, Ga.: I have had quite a little experience with ulceration of the cornea where there has been a fistulous condition left and where there is protrusion of the iris. In the South, where we have a great many colored people in the clinics, we have a great number of ulcerations of the cornea, and for the last six years I have been making use of conjunctival flaps with excellent results. It is quite common for these cases to come for protrusion of the iris, and

* Zeitschr. f. Augenheilk., 1914, xxxii, No. 6.

unless the process is stopped, the hernia becomes larger and the whole cornea becomes degenerated and lost. Under these circumstances I have had only one or two cases of fistulæ where I have been unsuccessful. My routine practice is to remove the protruding iris, make a conjunctival flap, and bring it over and attach it on the other side until it is adherent at the point where the iris had protruded, and I have been successful in more than ten cases.

DR. J. E. WEEKS, New York: I have had some experience in the Kuhnt operation for closure of fistula of the cornea which has been extremely satisfactory. The operation as practised by me has been almost identical with that described by Dr. Knapp, the fistula occurring near the center of the cornea, with the iris adherent near the margin. Iridectomy precedes the placing of the flap. The iridectomy is done and permitted to heal, or the iridectomy may be done at the same time. Before the flap is fastened in place the surface to be covered with the conjunctival flap has been denuded of epithelium and the fistulous tract thoroughly curetted. The healing in the cases that I have operated on has been perfectly smooth, and in the course of a month the greater part of the cornea, except the point of fistulization, has become almost clear, and the conjunctival flap has receded. The stitches were removed at the end of four or five days.

Recently I have had a case of dermoid tumor of the cornea in which, after removal of the tumor, the tissues of the cornea were so extremely thin that I have had to reinforce them by a conjunctival flap with two peduncles. In the case of the two conjunctival flaps, one is drawn across the cornea from one side and the other from the opposite side for the reinforcement of the cornea. The subsequent condition of the tissue has been most satisfactory.

THE AMBULANT AFTER-TREATMENT OF CATARACT EXTRACTION; WITH A NOTE ON POST-OPERATIVE DELIRIUM AND ON STRIPED KERATITIS.

HENRY DICKSON BRUNS, M.D.,
New Orleans, La.

For the last twenty-two years we have worked in the large clinic (about 3500 admissions annually) of a hospital with a limited housing capacity. The beds for white and for black, for men and for women, had, too, to be shared with the Ear, Nose, and Throat Department. Under these circumstances my colleague, Dr. E. A. Robin, and I have restricted our interned cases to the irreducible minimum. Thus we have had an opportunity to see how well the severest wounds of the eyeball will heal, if properly attended to, while the patient remains at home visiting the hospital daily for treatment and dressing.

In 1907 we were impressed by the case of a negro man who, having undergone extraction and the eye having been dressed, through a misunderstanding of orders walked to his home, some five miles away, and only returned for inspection forty-eight hours later. The eye being found in excellent condition, he was allowed to come and go in the same way until he had made an uneventful recovery. The restored vision was 20/40 with his first spheric glass.

We determined the next year, 1908, to treat a number of our patients after extraction as walking cases. In 1908 39 of our extraction cases were confined to the hospital and to bed. Of these 39 cases 5, or 12+ per cent., were failures: one through infection, one through iridocyclitis, one proved to have optic atrophy, and two injured their eyes

by striking them against the furniture or with their hands. Twenty-five were treated as ambulant cases; that is, the eye being dressed, they got down from the table and went to their homes afoot and in street-cars. Returning in twenty-four hours to the clinic, their eyes were redressed if there were complaints of pain; if there were none, the dressing was not removed for forty-eight hours. Of these 25 extractions, 2 were failures (8 per cent.), one infection and one iridocyclitis following discission of the after-cataract.

Reviewing all our extractions of senile cataract done in our clinic during the sixteen years from 1893 to 1908, both inclusive, we find that, counting the 39 cases in 1908, there were 371 cases confined to bed and to the hospital, with 39, or 10+ per cent., of failures. Counting the 25 in 1908, there were, during the six years up to 1915, 232 extractions of senile cataract with 18, or 7+ per cent., of failures among the ambulant cases. No case in which vitreous was lost was treated as an ambulant one.

For a complete understanding of the experiment I must be permitted to give a brief account of our practice. In the total 603 extractions, with 57, 9+ per cent., of failures, only cases of mature or immature senile cataract are included. The few hypermature cases are negligible. The complications, so far as we knew at the time of operation, were nothing worse than posterior synechiæ. From the beginning we have conformed our operation more and more strictly to the large corneal section in the limbus, with an iridectomy and with a moderate conjunctival flap; the capsule being opened with the point of the knife after puncture and before counterpuncture. In a number of cases we, especially my colleague, have used the long, undivided conjunctival bridge—with advantage, we believe, when loss of vitreous, infection, or perhaps other accidents are apprehended. The long undivided bridge, besides holding the edges of the incision together, keeps back the vitreous and

maintains unbroken the line of nutritive supply to a large part of the corneal flap. It is especially indicated in the extraction of dislocated lenses.

At many times during these twenty-three years, and before, the preliminary discission of immature cataracts has been tried, but always abandoned. We have made the extraction from five to thirty days after the discission, and once after twenty-four hours. The untoward uveal hyperemia induced more than offset any supposed advantage. During the prëambulant period, if I may be permitted the phrase, we made many simple extractions, but had abandoned this practice by 1908 as unsuited to hospital patients, save in exceptional cases.

Avulsion of a part of the capsule with Fuchs' forceps was also tried in a number of cases, but as we could see no lessening of the number of after-cataracts, we went back to opening the capsule with the knife-point. Complete anesthesia of the cornea and iris was obtained by deep post-ocular injection of the 10 (4 per cent. cocain), 10 (1 : 1000 adrenalin), 20 (normal salt) mixture (Robin, 1905).

Our patients being extraordinarily given to exploring the eye with their fingers, or to knocking it against the doors or the furniture, the dressing employed was directed to protection against such accidents. It was composed of a disc of gauze next the lids, a carefully disposed pad of absorbent cotton, a figure-of-8 bandage of the most elastic flannel, a cataract cage like a pair of wire tea-strainers joined together, the padded edges resting on the bones of the orbit, and over all a moist bandage of cheap mosquito-net containing much sizing, which set, when dry, into a light cast. For almost all of the twenty-three years the eye not operated upon was left undressed. While this dressing was effective in protecting the eye from blows and intruding fingers, striped keratitis was of almost constant occurrence.

About 1905 we adopted the dressing described by Pro-

fessor Blanco, of Madrid. It consists of a disc of fine gauze fastened with flexible collodion to the rim of the orbit and the inner side of the nose, a carefully placed pad of absorbent cotton, and over this another disc of gauze, slightly larger than the first, and also cemented down with collodion. Over this we put the cataract cage and the half figure-of-8 mosquito-net bandage. This is a lighter, cooler, and more exclusive dressing; but I mention its adoption particularly because, since its use, the striped keratitis with which we had been plagued has virtually disappeared. The only difference to which this can be ascribed is the pressure upon the eye produced by the former and the total absence of pressure by the Blanco dressing.

In the absence of pain, the dressing is never removed until forty-eight and sometimes seventy-two hours have passed. With lengthening experience our tendency is to leave the dressing longer and longer undisturbed, and, once removed, rarely and more rarely to renew it, but to cover the eye with the cataract cage only, under which the natural movements of the lids promote cleanliness and the riddance of secretions. Indeed, we have long been convinced that all dressings are, in the majority of cases, evils necessitated by the lack of perfect self-control on the part of our patients. Since 1908, when we began to enjoy the advantage of the examination by a skilled pathologist of the secretions of eyes about to be operated upon, we have, in a few cases in which the presence of pathogenic organisms could only be abated, or at best eliminated for brief intervals, done away with all dressings, allowed the patient to lie with the eyes closed, and instilled between the cautiously partly opened lids a 10 per cent. solution of argyrol at intervals of an hour, or as near thereto as possible. In each instance healing progressed without infection to a satisfactory result.

The after-treatment of all cases consisted in cleansing, with alternate instillations of 10 per cent. argyrol solution

and the borax-boric-acid-camphor-water wash, known in the clinic as "B. & C.," in a Todd's undine or irrigator; the use of atropin; and, if inflammation appeared, prolonged inunctions on the forehead, for relief of ciliary pain, of Arlt's salve (Ungt. hydg., 1 ounce; ext. belladon., 1 dram). Often carefully graduated amounts of dionin powder, to promote the absorption of cortical matter or exudative material, together with hot bathing of the closed eye, were used.

The cases were entirely unselected. So soon as the better eye was useless for reading the more mature cataract was extracted. The pursuit of this plan has inclined us to the belief that a previously healthy state of the eye to be operated on, especially a life-long freedom from any disease of the uvea, is more important in respect of a successful result than —within reasonable limits—the general condition of the patient.

At a meeting of the Section on Ophthalmology of the American Medical Association Dr. Walter R. Parker* read a paper on "Post-Cataract Extraction Delirium," with a report of 11 cases. He called attention to the fact that post-operative dementia was noted as early as the sixteenth century, that it is said to be more frequent after operations on the pelvic organs and upon cataracts, and he summarized the highly divergent opinions of the general surgeons upon the subject. In many respects the dementia following cataract extraction must differ from that sometimes following general surgical operations; for, even when infection takes place, the area is too small to give rise to those intoxications that beset more extensive operations. Cataract extraction, too, differs in one particular from almost all other surgical operations in that it is performed upon the aged only—the aged in fact, if not in years. I am not aware that delirium after discission of a juvenile cataract, or following the extraction of the lens for high myopia in a young

* Trans. Amer. Med. Assoc., Ophth. Sect., 1913, p. 395.

person, has ever been reported. The cases reported by Parker, and those mentioned by the members participating in the discussion, were all in old people; and it seemed to be the consensus of opinion that the dementia arose from the confinement in the dark under strange and, if one may say so, lonely conditions of an aged person. It seems to be fairly well established that dementia is more likely to occur when both eyes are bandaged, and that under such circumstances the removal of the bandage from one eye is almost sure to restore the reason.

Our experience with the ambulant after-treatment strongly confirms this view. During the time that we confined our patients to the hospital and to bed we had several (I am unable to state the exact number) cases of post-operative dementia. The delusion seemed to take the form of terror, or apprehension of great danger, and in not less than two instances the patients succeeded in eluding the attendants and in killing themselves by leaping over the bannisters or out of the window of the third story. Indeed, on reflection, there is nothing surprising in all this. All of us realize that old persons bear change badly; the time-worn machinery seems to keep going only because it can continue to run along the wonted grooves that have long since become those of least resistance. All, too, have observed that the effects, mental and physical, following an extraction are often entirely out of proportion to the severity of the operation. Hope and fear, dread and doubt, anticipation and resolve, all play a part. The old man's courage has been screwed up to the point of putting to the touch the most precious hope of the future, and when in a few minutes the unknown ordeal is over, what wonder that the tension drops so fast and far as to overturn the aged mind? The finer the nervous organization, or, in other cases, the greater the susceptibility of the ignorant and lowly to the dread of the unknown, the greater the pomp and circumstance of the operation, the

stranger, the darker, the stiller, the lonelier the after-treatment, the more likely is the mental disturbance to occur.

The power of these circumstances, and these alone, to bring about dementia after cataract extraction is attested by the fact that, among our 232 ambulant cases, not a single instance of this complication appeared. The risks and chances of the operation should never be made light of; it is one always to be done under the advantages of a modern operating-room; it should not be lightly undertaken either by the surgeon or the patient; but I cannot doubt that the comfort and *morale* of the patient are greatly enhanced by his prompt return to his home, his family, and the thousand and one particulars summed up in the phrase "familiar surroundings." There he should be enjoined to rest in bed or in a reclining chair and to observe those other precautions which medical common sense dictates to all convalescents.

It is needless to say that he should not visit the clinic unattended. The two instances only in which unfortunate results could without doubt be ascribed to the ambulant after-treatment happened through the neglect of this precaution. Two patients foolishly tried to reach the hospital alone; both had severe falls and their eyes were destroyed, the one by iridocyclitis, the other by an intraocular hemorrhage.

In conclusion let me say that I set no store by the small percentage shown in these 600 odd extractions in favor of the ambulant after-treatment; but it is at least interesting that the percentages are not markedly unequal.

DISCUSSION.

Dr. Arnold Knapp, New York: I would like to ask Dr. Bruns to explain the mixture used for the subconjunctival injection, and also to give us the particulars of the Blanco collodion dressing.

Dr. Bruns: Subconjunctival is rather a poor expression, and I corrected it in the paper when I looked it over. It is

really a deep post-bulbar injection. We use what we call the 10–10–20 mixture, and do nearly all our operations with it. It is a mixture of 10 drops of 4 per cent. cocain, ten drops of 1:1000 adrenalin, and 20 drops of normal salt solution, freshly and carefully prepared and injected with the hypodermatic syringe. This produces profound anesthesia; the iris can be cut without pain almost always. Contra-indications to its use for enucleation are tenderness of the eyeball, or youth or silliness of the patient.

The Blanco dressing has been a boon to me. It consists of two layers of gauze, the first fastened with collodion to the rim of the orbit just above the eyebrow, to the temple, and to the inner side of the nose; over this is placed a small pad of absorbent cotton; then a second piece of gauze a little larger than the first is placed over the absorbent cotton and cemented down also with collodion. The thing that interested me was the fact that when we used the flannel roller in an attempt to protect the eye, we would almost invariably have striped keratitis of a severe type, but since we have used the Blanco dressing I do not think I have seen more than one or two very mild striped keratitis cases. I have ascribed this to the fact that the former dressing produced pressure on the eye, and the Blanco bandage does not.

Dr. C. H. May, New York: Since discarding the bandage for cataract cases two or three years ago, I am sure that the patients have been more comfortable, that the healing has been much smoother, and that there have been fewer complications. I use a simple gauze and cotton dressing with a piece of adhesive plaster to keep this in place, the whole covered by a Ring mask. I am absolutely positive that the results have been more satisfactory all round since discarding the bandage.

I always inject a 5 per cent. solution of cocain five minutes in advance of the cataract extraction; and the injection is at the junction of the lid with the eyeball, as far as possible from the cornea, about two drops at the inner portion and two at the outer portion; this always gives satisfactory anesthesia.

Dr. J. E. Weeks, New York: I would like to ask Dr. Bruns what he means by 10 per cent. loss? If that is total loss, it seems to me to be a very high percentage.

In regard to subconjunctival injections, I would like to say that at the present time I practise this in all cases where the iris is to be attacked. The solution used is a solution of cocain, 1 or 2 per cent., and to that is added adrenalin (P. D. & Co. sol.) one part in ten, two or three drops being injected in two places. The injections are made ordinarily about 5 millimeters from the corneal margin. Operation three to five minutes after the cocain is injected.

DR. BRUNS: With regard to the percentage of loss, I admit that the figures are very poor. They are from hospital cases where we have to deal with every class of human beings, negroes, Italians, etc., people on whom most eye operations are unsatisfactory. The point is that by the same operator, with the two methods of after-treatment, the figures are about the same. Even if we had lost 50 per cent. under each plan, the point would be the same.

As to making the injections by placing two minims here and two minims there, etc., I do not believe there is any advantage in that whatever. You may as well take the syringeful, press the needle-point in the direction of the ciliary ganglion, and shoot the solution behind the eyeball, wait from five to eight minutes, and go ahead.

DR. E. C. ELLETT, Memphis, Tenn.: In regard to the occurrence of striped keratitis after cataract extraction, it occurred to me that it was due to the fact that the corneal incision was too small and the edges of the wound were subjected to an unusual amount of traumatism in the delivery of the lens.

I speak with diffidence about my personal experience, which is quite limited, but such as it is, since I began to do cataract extractions, I have seen a great variation in the occurrence of traumatic striped keratitis, and I have concluded that it is most frequent in those cases where the incision has been too small or in which the cataract has been immature, and due to the unusual amount of manipulation necessary to deliver the lens. However, it does not matter, because the cases all clear up anyhow.

In regard to the anesthesia, I have for many years used exactly the method suggested by Dr. Bruns, that is, the use of the 10–10–20 solution, with an interval of eight minutes

after injection, as he also advised, and it has been perfectly satisfactory.

Dr. R. A. Reeve, Toronto, Canada: Not having used the bandage in cataract extraction cases for at least twenty-five years, I have seen but few cases of striped keratitis, but I was not aware that exemption in these cases was due to any lack of pressure from the non-use of the bandage. My method has been to keep the eyelids in position by using isinglass silk plaster. The compress is fastened in position with two or three pieces of the plaster. I am indebted to Dr. Bruns for the explanation of the occurrence of striped keratitis, at any rate the coincidence is so marked that it strongly supports his contention.

Dr. Bruns: I merely desire to say in closing that, of course, I did not assume that this must be the only cause of striped keratitis, but evidently one important cause—pressure on the cornea. No doubt pressure on the cornea during an operation, for instance, when we have a long struggle to get a dislocated lens out, will cause it.

EXTRACTION OF CATARACTS IN THE CAPSULE BY A SLIGHT MODIFICATION OF THE VON GRAEFE METHOD.

ERVIN TÖRÖK, M.D.,
New York City.

The extraction of senile cataracts, being one of the most common and at the same time one of the most important operations in ophthalmology, has undergone a great many changes in its development. It developed slowly, step by step, from the several centuries old couching to the extracapsular extraction and then to the intracapsular extraction. Although this latter method has been known and practised by several surgeons for almost two centuries, it has not become of much importance until Smith, of India, described his, since well-known, method.

There is no doubt that the intracapsular extraction has its great advantages over the extracapsular method. The visual results in well-selected cases, *i. e.*, in cases where the fundus is normal, are always good, the vision being restored to normal, and there is no possibility of the formation of a secondary cataract; the eyes recover quicker, the post-operative complications, as iritis and infection, are less frequent, and, of course, entanglements of the capsule in the wound, with the often detrimental after-results, are excluded.

The method of Smith, according to his reports and to the reports of all those who have visited him and saw his operations, is excellent if the operation is performed by him or under his personal supervision and directions. The manipulations to dislocate and deliver the lens without serious complications require great skill and long experience. D. W. Greene* described these manipulations as follows:

"He (the operator) then makes light pressure with the point (of the delivery hook) on the lower border of the lens, backward toward the optic nerve. It is not possible to describe in words the amount and direction of this initial pressure, except to say, in a general way, that it should never be excessive or misdirected; one can only reach an appreciation of it by acquiring trained eyes and, best of all, an educated tactile sense. The purpose of this pressure is to dislocate the cataract, an entirely different series of manipulations being required to deliver it without accidents in a high percentage of cases, and these are difficult to describe. They should be learned at the elbow of a competent operator." Later, in discussing the disadvantages of the Smith method, he says: "The operation is highly technical, and can only be learned at the elbow of an expert who has plenty of material to demonstrate it."

This naturally means that the Smith method will never

* American Encyclopedia of Ophthalmology.

be accessible to the great bulk of ophthalmic surgeons. It requires an expert teacher and hundreds and hundreds of cataract extractions to master this "highly technical" operation. Very few ophthalmic surgeons are fortunate enough to get more than a few hundred cataract extractions in their lifetime, and in consequence the regular von Graefe extracapsular extraction will remain the routine cataract extraction until some easier and less dangerous intracapsular method has been found. Several ophthalmic surgeons have attempted this. Stanculeanu* has described a method of intracapsular extraction. He uses a special capsule forceps that have no teeth to tear the capsule. The forceps are introduced into the anterior chamber, a firm bite of the capsule is taken, and then, by movements from side to side and up and down, the zonular attachments are ruptured. The forceps are now removed and the lens is delivered, using two spoons, with one exerting continuous pressure over the cornea slightly below the center, and with the other one slight counterpressure is made above the wound. Vard Hulen† devised a vacuum extractor for the removal of the lens in its capsule. Knapp‡ modified the Smith intracapsular extraction by using the Kalt forceps for dislocating the lens and rupturing its zonular attachments, after which he delivers the lens according to Smith's method. I have witnessed many of Knapp's extractions and noticed what a firm grip the Kalt forceps take on the capsule, and how easily its zonular attachments can be ruptured with them. With the regular von Graefe method, if one uses a capsule forceps instead of a cystotome, it often happens with hypermature cataracts, where the capsule is thickened, that it does not rupture, but the zonule gives way and the lens is delivered in the capsule. This, of course, is an accidental

* Klin. Monatsbl. f. Augenheilk., 1912, p. 527.
† Trans. Amer. Med. Assoc., Sect. on Ophth., 1911, p. 122.
‡ Trans. Amer. Ophth. Soc., 1914, p. 666.

extraction in the capsule, but it happens not infrequently where a great many hypermature cataracts are extracted. I have tried to imitate this accidental intracapsular extraction by systematically using the Kalt forceps instead of the regular capsule forceps with teeth. The operation, therefore, with which I removed the 43 cataracts in the capsule is nothing else than a slight modification of the von Graefe extraction.

The patient is prepared for the operation in the usual way, a 1 per cent. solution of holocain being used for anesthesia. After the speculum has been inserted and the eye fixed, the puncture and counterpuncture are made in the limbus, and the incision carried up, following everywhere the limbus, and also completed there, securing at the same time a conjunctival flap. The incision, as in all intracapsular extractions, should be somewhat larger than the incision for a von Graefe extraction, encircling about one-half of the circumference of the cornea. After the regular small iridectomy, the pillars of the coloboma are replaced and the Kalt forceps are introduced into the anterior chamber with the left hand, and, while the right hand holds a Daviel spoon, the blades of the forceps are opened and with a light pressure backward a fold of the capsule is grasped. With a few lateral and circular movements the zonular attachments are loosened. Keeping the lens now firmly in hand with the forceps, the Daviel spoon is applied somewhat below the lower limbus. Both forceps and Daviel spoon are now manipulated at the same time. With the forceps we keep up these lateral movements, but at the same time a slight and gentle traction is exerted upon the lens forward, *i. e.*, in the direction of the anteroposterior axis of the eye. With the Daviel spoon a gentle intermittent pressure is applied to the sclera, *i. e.*, the sclera is slightly indented with the Daviel spoon and immediately released, then again indented and released. This is repeated several times, the motions of the

scleral wall being about the same as the motions of the wall of a pulsating artery. These manipulations usually rupture the zonule below, and the lower edge of the lens slowly emerges from behind the iris. The Kalt forceps now are slowly moved toward the wound, and the Daviel spoon, the gentle pressure with which becomes continuous the minute the zonule is ruptured, follows the lens, just as in the von Graefe extraction, and the lens is delivered with its lower edge first. The rest of the operation, *i. e.*, the replacement of the pillars of the iris and toilet, differs in no way from the usual von Graefe extraction.

The after-treatment is the same as used following a usual cataract extraction. I usually bandage both eyes for forty-eight hours, after which the non-operated eye is left open. The operated eye is bandaged for six or seven days, the dressing being changed and the eye washed daily; atropin is instilled according to necessity. All my patients sit up in bed twelve hours after operation, and in an armchair out of bed after twenty-four hours have passed.

I have tried this method of cataract extraction altogether in 53 cases. In 37 cases I have succeeded in removing the lens in the intact capsule; in 6 cases the capsule tore while the lens was engaged in the wound, and after the delivery of the lens it remained wedged in the wound in 4 cases and slipped back into the anterior chamber in 2 cases. In all these cases the capsule was removed afterward in its entirety by Kalt forceps. In the 2 cases where the capsule slipped back into the anterior chamber it was comparatively easy to reintroduce the Kalt forceps, get a hold of the capsule, and extract it without injury to the hyaloid membrane.

In 5 cases the capsule tore before the zonule of Zinn ruptured; a very large piece of the anterior capsule was removed, and the lens extracted in the usual (von Graefe) way. In one of these cases the lens became dislocated above, *i. e.*, the upper edge slipped under the upper edge of

the wound; the lens was easily replaced into its proper position with a spatula and then expressed in the usual way.

In 5 cases of immature cataracts I could not use the Kalt forceps, as they did not get a hold of the capsule but slipped off; the capsule forceps with teeth had to be used instead and the usual Graefe extraction performed.

I have lost vitreous in two cases, in none, however, during or in consequence of the operation. Both patients violently pressed the speculum against the eyeball just before its removal, after the completion of the toilet.

The recovery was, in most cases, quick and uneventful. In 4 I found a delayed closure of the wound. In these cases the eyes were irritated, the wound edges not well adapted, slightly gaping, and the conjunctival flap edematous. In five or six days the wound closed, and the recovery further on was uneventful. I cannot explain this delayed closure, since the pillars of the iris were free; it is not impossible, however, that, since the upper edge of the lens is delivered last, some of the fibers of the zonule were pulled up and wedged into the wound.

In one case a severe iridocyclitis with plastic exudate developed on the fourth day after the operation, resulting in a total occlusion of the pupil and coloboma, with perfect perception of light and localization.

In one case the day after the operation we found the eye red and irritated and considerable blood in the anterior chamber; the blood absorbed slowly and the eye recovered perfectly, but in the pupillary area the hyaloid membrane seemed to be somewhat elevated above the level of the iris, and this cyst-like elevation contained some blood. The patient saw 20/40 with the proper correction, but complained of a constant blur before the eyes. We have watched this condition for nearly two years, but the blood has not absorbed entirely. The same patient's other eye was operated on, and the lens removed in the capsule about

one year after the first operation, and the same curious condition developed. A similar condition developed in another case where only one eye was operated on, the vision being only 20/70 with correction. The visual results in the 43 operations where the lens was removed with its capsule are the following (the 6 cases are included where the capsule ruptured, but was removed entirely afterward):

20/30 or better..... 34 cases
20/40..... 2 cases

These are two eyes where blood under the hyaloid membrane in the pupillary area was found.

20/50..... 1 case

In this case much blood was found in the anterior chamber following the operation, and considerable amount of blood-pigment was left on the hyaloid membrane.

20/70..... 3 cases

In two of these cases there were opacities in the vitreous following loss of vitreous, and in 1 case there was blood under the hyaloid in the pupillary area.

20/100..... 1 case

This was an amblyopic and strabotic eye.

6/200..... 1 case

In this case, after the removal of the cataract, we found central choroiditis in the fundus.

Hand movement..... 1 case

In this case a plastic iridocyclitis developed following the operation.

In conclusion I wish to say that in my limited experience the removal of the lens in its capsule with this slight modification of the von Graefe method is very simple and easy for any one who is experienced in the usual von Graefe extraction. I believe that the danger of loss of vitreous is even

less than in the usual von Graefe extraction, because the lens is removed by a combination of traction and pressure, and the pressure to be applied is infinitely less than that used in the former method. The Kalt forceps are very well adapted for use in mature, hypermature, and nuclear cataracts; they can, however, not be used in immature cataracts. In these latter cases I believe they could be substituted for to an advantage by Vard Hulen's suction extractor or some similar device.

IMPROVED CAPSULE FORCEPS FOR INTRACAPSULAR CATARACT EXTRACTIONS.

F. H. VERHOEFF, M.D.,
Boston, Mass.

Two years ago Arnold Knapp described before this Society a new method of intracapsular cataract extraction which he and Stanculeano had independently devised. In this method the lens-capsule was grasped with Kalt forceps and the lens dislocated by moving it from side to side, after which the forceps were removed and the lens expressed as in the Smith method. About a year ago I was informed that Török had modified Knapp's method in the following way: Instead of dislocating the lens he retained hold of the latter with the forceps and gently pulled it out while at the same time he made pressure on the cornea in the usual manner. Török's modification impressed me so favorably that I immediately tried it and have since continued to use it in almost all of my extractions of senile cataract.*

I have found that the method is most successful in cases of

* Since this was written I find Török dislocates the lens by Knapp's method before withdrawing it. Török's method is therefore quite different from mine. I am surprised to learn also that Török does not regard his method suitable for immature cataracts.

immature and hypermature cataracts; in the former evidently because the capsule is not degenerated, and in the latter because the capsule has become thickened and the zonule weakened. Lenses showing diffuse central haziness without marked peripheral striæ are especially suitable for the operation. Congenital cataracts in adults over thirty years of age I have also found may be safely removed by this method, for, although the zonule is apt to be resistant, the capsule is still more so. According to Smith, congenital cataracts are not suitable for his method of intracapsular extraction.

In regard to the question as to the advisability of adopting the method as a routine procedure in all cases of mature senile cataract I have not yet come to a definite opinion. In some cases of mature cataract the lens comes away readily with the capsule intact, but in many the capsule ruptures, and in these cases I think the danger of loss of vitreous is somewhat increased. In the only case of intumescent cataract in which I tried the method the lens came out intact readily enough, but was followed by a slight amount of vitreous. In cases of immature cataract the danger of loss of vitreous is negligible, especially when compared with the advantages to be gained by removing the lens in capsule.

Whether or not I have carried out the operation in all its details in the same way as Török I do not know, since previous to this meeting he has not published a description of his method. I have found it necessary to make the incision only slightly larger than that for the ordinary method, since the lens tends to mold itself to the opening. It is important, I think, to grasp the capsule as low down as possible, so that when the lens is withdrawn traction will be made upon only a few fibers of the zonule at a time and less resistance thus encountered. For this reason, as well as others, I dilate the pupil with atropin previous to the operation. After the capsule is grasped, the first movement should be to

bring the lens directly forward toward the cornea; then it should be gently pulled out while deep pressure is being made along its lower edge through the cornea by means of the point of a large strabismus hook.

In most of my cases the operation was performed with iridectomy and without a conjunctival flap. During the past six months, however, I have employed a new method of wound closure which has enabled me safely to leave a round pupil. This method, which will be more fully described later, is briefly as follows: Before the cataract incision is made, a small quadrilateral conjunctival flap is dissected up above the cornea. A double-armed suture is then passed superficially through the sclera for about 1 mm. parallel to and close to the limbus beneath the flap. Each needle is then passed through the flap, leaving two loops of thread to allow room for passage of the lens. The two loops are then drawn to the outer side so that the cataract knife cannot cut them. An ordinary cataract incision is now made through the limbus, care being taken to bring the knife out beneath the conjunctival flap and in front of the scleral suture. If for any reason the anterior chamber is lost while the incision is being made, and the iris prolapses in front of the knife, the latter should be allowed to remain in position and the chamber refilled by inserting the tip of the irrigator into the incision along the blade. The lens is then removed as described, the iris partly replaced, and the suture firmly tied with a surgeon's knot. The iris is now completely replaced and then two very small peripheral buttonholes are made in it, one on each side of the conjunctival flap. When the capsule ruptures, or in case of an ordinary extraction, cortical matter is removed by irrigation after the suture is tied. The closure of the wound is so perfect that on one occasion I permanently restored the anterior chamber by means of the irrigator immediately after the operation. It probably would not be wise to do this in all cases, however, because

should the patient break open the wound the sudden gush of aqueous might cause prolapse of the iris. This method of wound closure I believe to be an almost certain safeguard against infection subsequent to the operation.

After having had considerable experience with the Kalt forceps in intracapsular extractions, it occurred to me that they could be improved in several important respects. In the first place, I found that it was not an easy matter to open the blades just to the proper extent and at the same time make the correct amount of backward pressure in the act of grasping the capsule. This difficulty I have overcome by means of a stop which allows the forceps to open only to the proper distance, about 2.5 mm. This stop also acts in such a way when the forceps are closed as to limit the

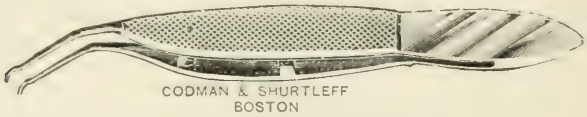


Fig. 1.—Improved capsule forceps.

amount of pinching pressure to which the capsule can be subjected. In other words, with this modification the only delicacy of manipulation required in grasping the capsule is in making the backward pressure upon the lens. Another serious defect in the Kalt forceps is that they are especially narrow just at the place where they must be held with the fingers. This, together with the facts that they are unduly top-heavy and not corrugated, makes them difficult to manipulate. I found also that the jaws of the Kalt forceps were bent at far too great an angle. These defects I have attempted to overcome in the forceps here illustrated (Fig. 1), and in addition I have modified the biting ends of the instrument. Another improvement consists in the fact that the blades of the new forceps can come in contact only at the biting ends and at the heels, a space being left be-

tween these points which, in case of a simple extraction, renders it impossible for the iris to be caught. The instrument was made for me by Codman & Shurtleff, of Boston.

DISCUSSION OF PAPERS OF DRS. TÖRÖK AND VERHOEFF.

DR. CARL FISHER, Rochester, Minn.: About a year ago I had the privilege of seeing Dr. Knapp do some of these operations by the method that he described (not by the Török method), and it impressed me as being a beautiful and safe way of making the extraction of the cataract in the capsule. Since then I have been trying to do the operation as nearly as Dr. Knapp does it as possible, with the exception that where I have not been able to grasp the capsule easily, I have not forced the procedure. I have had only a small series of eleven cases, and in all but two good results with vision over 20/30. In two of the cases there was a bulging of the vitreous from the wound, not at the time of operation but during convalescence, and it occurred to me, as Dr. Verhoeff described his modification, that this would obviate this complication. In all of the other cases I had exceptionally beautiful results.

DR. W. B. LANCASTER, Boston, Mass.: It seems to me that the method of Dr. Török is a great improvement over any other method of intracapsular extraction. I have tried it on a few cases, but have not had the opportunity of seeing it done by the inventor.

In regard to using it in cases of immature cataract, I am not so pessimistic as Dr. Török, though I agree with him that there is difficulty in some cases in grasping the capsule. It seems to me that it has helped me to modify Dr. Török's method in this way: Instead of waiting until after the capsule is grasped before applying counterpressure, apply the counterpressure simultaneously with the grasping of the capsule. This pressure supports the lens from behind so that you can press the capsule forceps more firmly on the anterior surface of the lens without premature rupture of the suspensory ligament and loss of vitreous. It is obviously necessary to cause the jaws of the forceps to make dents in the surface of the lens, raising a mound between them which

becomes a fold when the forceps are closed. I have been surprised to see when the lens came out in the capsule that the consistency of the cortex and character of the capsule were such as to make the grasp of it easy. When you take the lens in your hand after it is out, you find the capsule slack enough and the cortex soft enough so that you can pick up a fold and get a firm grasp upon it without tearing it. I had erroneously conceived of the capsule as kept smoothly stretched, plumply filled, by the lens substance. I was surprised to find, on the contrary, that in those which I examined the capsule was sufficiently redundant to permit easy raising of a fold by pressing the separated jaws of the forceps upon the capsule, depressing it into the soft cortex, and closing them. Instead of saying the capsule was redundant I should have said perhaps that the lens was sufficiently shrunken to make the capsule relatively lax or redundant. In some immature swollen cataracts this is probably not the case, and it may be that these are the ones in which Dr. Török has found his method unsuitable. I have seen an operator try unsuccessfully to grasp the capsule of a fresh traumatic cataract with a view to its extraction in this manner—of course, the lens is swollen in such a condition. It is extremely difficult to grasp the capsule of a fresh pig's crystalline even with toothed capsule forceps, simply because the capsule is so smooth, plump, and tense.

It has seemed to me that there are more opacities in the vitreous immediately following the intracapsular operation, and I should like to inquire whether that is a coincidence which has happened in the few cases I have seen, or whether it is, perhaps, some effect on the ciliary body that gives rise to a little exudate that makes a slight opacity in the anterior vitreous.

One great advantage of the method seems to me to be that you are not necessarily committed to extraction in the capsule even after you have started to operate in that way; thus, if you do not get a grasp on the capsule and dislocate the lens, you can relinquish the intracapsular plan, do a capsulotomy, and extract in the usual way. I have done this in three cases.

THE ETIOLOGY OF IRITIS.*

E. V. L. BROWN, M.D.,
Chicago,

AND

ERNEST E. IRONS, M.D.,
(By invitation)
Chicago.

This study was undertaken primarily to determine the causes of "rheumatic" iritis, but in the absence of any clear distinguishing features of this form, apart from etiology, we have included all cases of non-traumatic iritis,† and, after examining each patient as completely as possible, have grouped them in accordance with the causes discovered in each.

This report does not assume to furnish a complete catalog of the causes of iritis, but is rather a recital of what infections we found and of the evidence for and against the etiologic relation of each to the iritis in the patients we have observed. The final conclusion as to the relative importance of each cause must be left to future studies.

One hundred patients (47 private and 53 clinical) have been thus studied.‡ Careful attention was given to the history, and a complete physical examination was made to

* From the Memorial Institute for Infectious Diseases and the Presbyterian and Cook County Hospitals.

† The eye condition met with has been, for the most part, "plastic" iritis. Certain cases showed a more marked involvement of the ciliary body or anterior choroidea, and the diagnosis was accordingly modified to "serous" cyclitis or to iridochoroiditis. All cases were studied during an active stage of iritis. No cases of sympathetic iritis or iritis following wounds, operations, or ulcers have been included. We have not encountered any cases of fulminating suppurative panophthalmitis during the period of this study.

‡ We are indebted to Drs. Wescott, Pusey, Wilder, Darling, Beard, and Blue for the privilege of using the notes of cases referred to one of us (E. E. I.) for study. We are also indebted to a number of our colleagues, whose coöperation in painstaking and laborious clinical and laboratory examinations has added much to the completeness and accuracy of our study. Among others, we acknowledge the valuable assistance of Drs. Ormsby, Harris, McEwen, and Stil-

detect the presence of syphilis, tuberculosis, gonococcal infection, and infections of teeth, tonsils, sinuses, prostate, pelvis, or other structures which might give rise to lesions in joints or eyes. Laboratory examinations included Wassermann tests (98 patients) controlled by two laboratories, complement-fixation tests for gonococcal infection, radiographs of teeth and sinuses and of the lungs, where there was any question of pulmonary disease; cultures of pus from tonsils, sinuses, prostate, and other infected tissues. Tuberculin tests, using 1, 3, 5, 10 mgm., were made in suitable cases.

After a complete survey of the patient, appropriate measures were taken to remove all infections, so far as this was possible. From the course of the iritis following these procedures we obtained valuable evidence confirmatory of the etiologic diagnosis. In patients in whom we found but one source of infection, removal of which was followed by immediate improvement of a previously intractable iritis, no special difficulty was experienced in drawing reasonably safe conclusions as to the relation of cause and effect.

We found two or more infections associated in a surprisingly large number of patients. In many of these the reason of the association was obvious; thus, infections of tonsils, sinuses, and teeth, or syphilis and gonococcal infection were often found together. In other instances there was no apparent direct relation between two infections, *e. g.*, syphilitic iritis from secondary syphilis associated with a latent alveolar abscess.

By a careful weighing of all the evidence we were able to determine, in most cases, which infection was responsible for the disease of the eye, but sometimes the available evidence was insufficient and we have placed such cases in a

lians in the diagnosis of certain cases of syphilis; Drs. Shambaugh, Friedberg, Boot, Beck, and Sonnenschein in examinations of the nose, throat, and sinuses; Drs. Gilmer, Moorehead, Potts, Puterbaugh, and McGuire for advice in and treatment of dental conditions; Drs. Potter and Blaine for many radiographic studies of teeth and sinuses; Drs. Nicoll, Bissell, and Moody for extensive serologic studies.

"combined" group. Some of these patients suffered from several infections. Syphilis, active tuberculosis, gonococcal infection, and alveolar abscess were repeatedly found in the same individual.

Table I summarizes—(1) The number of times each infection was found in direct etiologic relation with the iritis; and (2) the coincident infections found.

TABLE I.—THE CAUSES OF IRITIS IN 100 CASES.

	Alone	With Other Infections	Total	Coincident Infections						
				Syph.	Gon.	Tbc.	Dental	Tonsil	Sinus	Genito-urinary
Syphilis.....	10	13	23	..	8	5	5	1	1	..
Gonococcal infection	7	2	9	..	..	..	1	1	..	..
Tuberculosis...	8	..	8	..	..	..	..	..	..	..
Dental infection.....	7	11	18	2	2	2	..	7	1	..
Tonsillar infection.....	7	9	16	1	3	2	7	..	..	..
Sinus infection	1	2	3	..	..	..	1	1	..	..
Genito-urinary (non-ven.)...	3	..	3	..	..	..	..	..	..	..
Other infections	2	..	2	..	..	..	1	..	..	..
No cause found	1	..	1	..	..	..	..	..	..	..
Combined infections.....	..	..	17	8	9	7	8	13	5	1
			100							

In the above table syphilis appears as the cause of the iritis in 23 cases, and in 11 other instances appears under coincident infections, a total of 34. In five other cases there was some reason to think that syphilis should be considered either because of an old history of infection in two cases (twenty and forty-five years previously), with repeated negative Wassermann tests and no other evidence of syphilis; in two cases a 1 + Wassermann test without history or other evidence of syphilis; in one case a doubtful history of infection three years previously, for which vigorous treatment was given, with at present no other evidence of syphilis and repeated negative Wassermann tests. In all five there were other active infections present; in one, dental; in one, sinus; in one, tonsillar; in one, gonococcal; and in one combined infection of sinuses and tonsils. In none of these five cases did the evidence seem to warrant the consideration of syphilis as a coincident infection in the same sense that the term was used in the table for other cases in which syphilis or other infections were coincident, but in the discussion of syphilis as a cause of iritis (see below) we did not feel justified in omitting any cases in which syphilis could in any respect demand consideration even though the evidence was doubtful, such as a + 1 Wassermann, and so these five cases are included with the 34 noted in the table, making a total of 39 noted in the text.

Syphilis.

The relation of syphilis to iritis affords one of the most interesting studies in this series.

The history of infection and the evidence of past and present infection were carefully investigated in each case. In 98 cases Wassermann tests, usually controlled by two laboratories, and in doubtful cases repeated two and three times, were made. In the 2 cases in which this test was not made there was no history or clinical evidence of syphilis, and the iritis in both was definitely ascertained due to other causes.

In 39 cases syphilitic infection had occurred either recently or years before. Of these, 28 were dispensary and 11 private patients. In 20 of the 39 cases the iritis was clearly syphilitic; of these 20, 13 showed other clinical evidence of active syphilis. Of the 7 in which other active syphilis was not apparent the Wassermann was positive in all. The iritis was recurrent and had been previously observed in association with acute syphilis in 4; a condyloma of the iris was observed in another; in 2 the diagnosis would have remained in doubt without the Wassermann test. All 7 recovered promptly on antiluetic treatment. Three other cases of the 39 probably were due to syphilis, judging from the prompt relief following antiluetic treatment, although other possible causes, such as active gonococcal or tonsillar infection, were also present.

The remaining 16 cases of the 39 are of interest because they illustrate the obvious fact that a person infected with syphilis may also suffer from bacterial infections, which may be the real cause of secondary metastatic lesions. We shall refer later to the influence of one infection in the body on other subsequent infections.

Of the 16 cases in which the iritis was not syphilitic, the grounds on which syphilis was excluded as the cause varied.

In none were there signs of active syphilis. In 5 the time of infection was remote—seven, twenty-two, twenty-five, twenty-seven, and forty-five years previously, and the Wassermann negative. In all there were other causes present, following the removal or appropriate treatment of which, where this could be done, the iritis subsided without antiluetic treatment. In several of these the iritis had previously resisted antiluetic treatment in other hands. Among the causes of the iritis in these cases were tonsillar, sinus, and gonococcal infection with arthritis. In 3 the iritis subsided at once after the drainage of alveolar abscesses. In 1 case, due to alveolar abscesses, the iritis appeared contemporaneously with extensive arthritis, while the patient was receiving mercury for syphilis of twenty years' standing. Removal of the abscesses resulted in prompt cure of arthritis and iritis.

The distribution of syphilitic iritis between the dispensary group and the private group of cases is of interest. Of 47 private cases, syphilis was present in 11 and was the cause of the iritis in 6, while of 53 cases in the clinic, syphilis was present in 28, and was the cause of the iritis in 17. The cases in the clinic at Cook County Hospital were in part referred to us as out-patients, and in part came from the wards of the County Hospital, including the venereal ward. The inclusion of several cases of syphilitic iritis from the venereal ward raises the proportion somewhat above what might be encountered in a hospital which does not receive active syphilis. We have been able to discover no other reasons for assuming that the figures given do not fairly represent the proportion of syphilitic iritis in a metropolitan clinic, so far as conclusions may be drawn from a limited number of cases.

In unselected groups of cases syphilis is encountered more frequently in the wards of a charity hospital than in private practice, and in the latter also the disease is more likely to

be efficiently treated, and the complications of untreated syphilis are therefore less frequent.

Out of 100 successive cases of iritis, there were only 23 in which syphilis was the cause of iritis, and 16 others due to other causes in which syphilitic infection had occurred, making a total of 39 patients in whom syphilis had to be seriously considered in the etiologic diagnosis. This leaves 61 cases in which a searching examination failed to reveal any evidence of past or present syphilitic infection, and in which, therefore, the iritis could not be reasonably thought to be due to syphilis.

So far as one may draw conclusions from this number of cases, it would seem that the widely accepted statement that 50 per cent. or more of iritis is due to syphilis may have to be revised. Certainly in the absence of other evidence of active syphilis in a patient with iritis the assumption that the iritis is syphilitic is more likely to be wrong than right.

DENTAL INFECTION.

Infections about the teeth occur in a large proportion of all persons, and care must be exercised in ascribing to such infections a causal rôle when they happen to be found in the subjects of iritis or other metastatic lesions.

In this study we have not included peridental infections as possible causes, unless they were closed processes, *i. e.*, apical infections (alveolar abscesses), demonstrated by radiographs and confirmed by the oral surgeon on extraction.

In certain cases of extensive pyorrhea lateral abscesses occur, or the tissues may be deeply invaded, with resulting metastatic lesions, but in the present series such cases have not been encountered. In several cases alveolar abscess was the only discoverable infectious process in the body, and the removal of the abscess was followed by prompt and permanent relief of the iritis.

Alveolar abscesses, or apical infections demonstrated by

radiographs and confirmed by the oral surgeon, were found in 41 patients (24 clinical and 17 private). In 18 patients (10 private and 8 clinical) we regarded the dental lesion as the source of the iritis. The evidence on which this conclusion was based varied in the several cases, but included such facts as the absence of other sources of infection and the prompt clearing of the eye on removal of the cause, or the association of the initial attack or recurrences of iritis with known dental infection, or, if other infectious processes were also present, the failure of treatment to relieve the iritis until the abscesses were discovered and removed.

In 10 clinical and 5 private patients the iritis seemed to be clearly due to other causes, such as syphilis, tonsillar infection, etc., and the dental infection relatively inactive so far as concerned the causation of metastatic lesions. Latent alveolar abscesses are often found in persons not suffering from iritis or other evident metastatic disease.

In the remaining 9 patients (7 clinical and 2 private) the dental infection was associated with other infections, often two or more, including tonsillar, sinus, syphilitic, or gonococcal, and we were unable to satisfy ourselves which infection was responsible for the disease of the eye. So far as possible we tried to arrange the sequence of surgical and other treatment so that we could observe the effect of each procedure on the eye, but in a number of instances considerations of expediency and the saving of vision made it necessary to proceed rapidly to the elimination of all possible causes of the iritis.

Cases of this type, in which the difficulty in assigning a cause for the iritis arose not from inability to find an infectious process, but in deciding between several adequate causes we have preferred to group under the heading of "combined infections," rather than to guess as to which of the infections was the exciting cause of the iritis. This group will be discussed later.

Tonsillar Infection.

Tonsillar infections appear to be as important an etiologic factor in iritis as in arthritis and other metastatic lesions. We have included under tonsillar infections deep infections of the tonsils, intratonsillar and retrotonsillar abscesses, but have not included slight lesions, such as small crypts containing "cheesy" material, unless accompanied by deeper infection. Superficial infections are not to be disregarded in the routine examination, however, for they often furnish a clue which leads to further investigation and the discovery of deeper hidden infection.

In some cases the history of previous tonsillar infections, the appearance of the tonsils, the enlargement of regional lymph-nodes, and the associated arthritis and myositis, point directly to the tonsil as the source of the iritis, but in other cases careful and repeated search is required to discover the deep tonsillar abscess, the removal of which results in relief of the iritis.

The tonsil was clearly the source of infection in 16 cases (12 private and 4 clinical). In 13 cases of the combined group tonsillar infection was present with other infections, but, as in the case of dental infections, we were not able to determine whether tonsillar or dental infection was the immediate source of the iritis. It is of some interest to note that 12 of these 13 combined cases were in the dispensary group, and if all the facts could be determined it is probable that a number of these would fall in the tonsillar group, thus raising the proportion of tonsillar cases in the dispensary group from 4 clearly defined cases to a number more nearly corresponding to that in the private group (12 cases), in which multiple infections were less numerous, and consequently an etiologic diagnosis more easily made.

In cases clearly due to other causes tonsillar infection was found in several; in the syphilitic group 2, gonococcal 1,

sinus 1, dental 7. Tonsillar and dental infections are frequently found together, and the question may well be raised as to whether the dental infection may not have developed in previous months and years by the same mechanism as the iritis, by repeated infections from the tonsils. Cultures from tonsillar abscesses after tonsillectomy usually showed hemolytic streptococci.

GONOCOCCAL INFECTION.

Gonococcal infection was believed to be the cause of the iritis in 9 patients, all males. In 8 of these an active or recent, usually multiple, arthritis was present. In 7 the iritis was recurrent and often associated with recurrent arthritis following exacerbations of urethritis. In 1 case the iritis occurred during an acute gonorrhea without arthritis. In another a recurrence of the iritis was noted during a period of fever, with arthritis, and the gonococcus was isolated from the blood. A positive gonococcal complement-fixation test was obtained in 6 of 7 cases in which it was made. Three of these patients came to one of us for advice as to arthritis, so that it is possible that the proportion of gonococcal iritis is slightly larger than would be the case in a second 100 cases in which this element of selection was not operative.

In 7 of the 9 patients infections other than gonococcal were not found; in the other 2, both recurrent, tonsillar or dental infections were found, but the recurrences were closely associated with exacerbations of urethritis and arthritis. It is, of course, conceivable that a urethritis with general infection and arthritis might so depress the resistance of a patient to other infections that a latent infection in the tonsil might give rise to an iritis, but such an assumption seemed hardly justified in the two cases under discussion. All evidence in each case must be considered, however, for the mere finding of a positive complement-fixation test in the subject of gonorrhea does not indicate a gonococcal origin

of the iritis, any more than a positive general tuberculin test alone indicates that an iritis is tuberculous.

In cases due to other causes gonococcal infection was present in 13 : 8 of the syphilitic group, 2 of the dental, and 3 of the tonsillar, all dispensary patients.

In the combined or undetermined group evidence of active gonococcal infection was found in 9, of whom 8 gave a positive complement-fixation test. All 9 patients were in the dispensary group. One or more attacks of multiple arthritis with recurrences of urethritis had occurred in several of these patients. In 1 patient who had contracted syphilis twenty-two years previously the initial attack of iritis occurred four years later coincidentally with arthritis and directly following a urethritis. During the year preceding the present attack the iritis had recurred coincidentally with the appearance of tertiary syphilitic lesions of the leg and mouth. At the time of examination the syphilis had healed under treatment, but the iritis was still active, the Wassermann was negative, gonococcus complement fixation positive. The mouth was extremely foul, with extensive caries and gingivitis, apparently not mercurial, but no alveolar abscesses were found. A positive tuberculin reaction followed the injection of 1 mgm. O. T., and later an acute attack of tonsillitis occurred. Here it seems reasonable to assume that the initial iritis may have been gonococcal, but the possibility of the active participation of one of the other coincident infections cannot be excluded.

Such cases as the above illustrate the difficulty of evaluating the several possible causes of iritis in patients, especially of the dispensary group, in whom multiple infections are frequently found.

Tuberculosis.

In 8 patients (3 clinical and 5 private) the iritis appeared to be tuberculous. In 7 the evidence seemed clear, and in 1 presumptive. In 2 of the 7 active clinical tuberculosis was

present, and in 5 positive tuberculin reactions, together with other evidence of tuberculosis, such as changes in the lungs, were demonstrated by physical examination and radiographs. In 2 cases focal reactions in the eye repeatedly occurred following tuberculin tests; in interpreting these reactions care was taken to avoid confusing them with the sudden exacerbations of inflammation which may occur spontaneously in iritis. In none of the 7 was there any other discoverable focus of infection.

Evidence of the presence of tuberculosis, usually latent, was found also in 5 patients in whom the iritis was due to syphilis; in 2 each of the dental and tonsillar groups; and in 7 of the combined group, the latter all dispensary patients.

SINUS INFECTION.

In 3 patients the iritis was considered due to infection of the sinuses. In several others the sinuses were infected together with other structures, such as tonsils and teeth, but it was not possible to determine whether one or another of the lesions was the immediate source of the iritis, and these cases were accordingly placed in the combined group. For practical purposes the importance to the patient lies in the recognition of the presence of infection, whether it is in one or more places, and its eradication.

Abnormalities in the several sinuses were discovered on routine clinical examination or by radiographs in 19 cases, in 10 of which active infection was demonstrated. In spite of able assistance and the recent advances in radiographic and clinical diagnosis of sinus disease, we have experienced more difficulty in determining the presence or absence of infection in the sinuses than in any other region of the body.

OTHER INFECTIONS.

In 3 cases the iritis was associated with non-venereal in-

fections of the genito-urinary tract, including 2 of the prostate.

In one instance no adequate cause for the iritis was found.

Diabetes was present in 3 cases, but in each instance infections were also present—in 1, alveolar abscess; in 1, tonsillar abscess; and in 1, tuberculosis, syphilis, and tonsillar infection.

Several cases came to us with a provisional diagnosis of "iritis from autointoxication." In none of these did we fail to find active infectious processes, such as tonsillar or alveolar abscess, and the previously resistant iritis subsided on relief of the infection without specially directed treatment of the supposed "gastro-intestinal autointoxication."

When anemia, weakness, anorexia, achylia, diarrhea, or constipation occurs in the course of an acute infectious disease, or in certain chronic but clinically evident infections, we recognize that they are the results of disturbance of the functions of the body arising from the infection, but when they are associated with unrecognized infections, together with metastatic lesions of the joints or eyes, we may be tempted to correlate the gastro-intestinal disturbance with the arthritis or iritis, and overlook their common cause. There is no question as to the importance of the regulation of diet, of the correction of constipation, and of all other measures which will improve nutrition and increase bodily resistance to infection, but even these important procedures should not be allowed to supplant or interfere with the search for and the eradication of infectious foci.

Combined Infections.

There were 17 patients in whom several infections were present, or in whom there were several foci harboring the same or different infections in such combination that we could not with certainty determine which infection had given rise to the iritis. These cases have already been

referred to in discussing the incidence of the several types of infection.

The patients belonged largely to the dispensary group, and careless habits of life and the physical handicap imposed by the disability arising from some one infection, such, for example, as gonococcal arthritis or pulmonary tuberculosis, no doubt contributed to the lessening of resistance and made the sufferers more susceptible to subsequent infections. The unfavorable action as regards the host of one infection upon another was probably also a factor in producing the large number of infections and their complications in this group.

But even in this unfavorable residue the results of treatment directed toward the elimination of the infections were remarkably good, and we had the satisfaction of seeing many hitherto stubborn cases of iritis relieved.

In 12 of the 17 recovery followed the removal of infectious processes. In 2 cases satisfactory results were not obtained despite months of treatment; secondary glaucoma was present in both.

In 2 we were unable to complete the eradication of infections, and 1 case is improving slowly under tuberculin.

THE MECHANISM OF THE PRODUCTION OF IRITIS.

Focal infections are very frequent, and yet many persons do not suffer from iritis or other metastatic lesions. Sepsis, streptococcal or pneumococcal, is also frequent, but metastatic lesions of the eye are relatively rare. Given an alveolar or tonsillar abscess, what are the causes which lead to the production of iritis or arthritis in one person and not in another, at one time and not at another? An adequate answer to this question requires a more complete knowledge of the mechanism of immunity than we now possess, and certainly a discussion of what is already known would require more space than is at our disposal. A consideration of some of the factors involved is possible, however.

Iritis can be produced in animals by intravenous injection of bacteria, as shown by Stock and others.

Bacteria reach the eye by the circulating blood, and can be recovered from the eyes of animals in which iritis has been experimentally produced.

In some recent experiments we have produced iritis in rabbits by intravenous injection of streptococcus, Bacillus mucosus capsulatus (Friedländer), Bacillus pyocyaneus, and gonococcus. Usually, however, in order to accomplish this large doses must be given, so that a condition of sepsis is created, and then only in occasional animals is iritis produced. Certain of these animals recover, and the lesion of the eye heals.

If one eye of each of a series of rabbits is injured by intraocular injection of minute amounts of dead bacteria, and then, after the eyes have healed and all active inflammation has disappeared, an intravenous injection of living bacteria is given, there follows an inflammation of a larger proportion of the injured eyes than of the control eyes. By this procedure we have produced ocular inflammations by the injection of staphylococci, as well as streptococci.

Such experiments bear directly on the problem of recurrences of iritis.

In addition to the factors of sepsis, fortuitous embolism, and previous trauma to the eye, changes in the organisms themselves must be considered in a study of the causes which determine the occurrence of lesions of the eye. In a case of streptococcal infection of the tear-sac which had presumably given rise to iritis we were able to produce iritis in rabbits by injection of streptococci when first isolated from the patient.* Later these same cultures and other cultures obtained at subsequent dates from the patient failed entirely to produce lesions of the eye. We were not able to

* "The Localization of Streptococci in the Eye," Jour. Infect. Dis., 1916, xviii, 315.

make out any changes in morphology or cultural reactions coincident with the change in localization with respect to the tissues of the eye.

From this clinical study, and from our experiments thus far, it would appear that not one but several factors are concerned in the production of iritis, among which may be noted the presence of a focus of infection in which bacteria may live and multiply, partially protected from the resisting forces of the host; the periodic entrance into the blood-stream of these organisms and their lodgment in the eye, either by reason of their great number, as in sepsis, or by fortuitous embolism; changes in the eye itself, either in vascular anastomoses or in susceptibility of the ocular tissues, owing to previous injury; and, finally, changes in the organism itself, whereby it may become better able for a time to maintain itself in the tissues of the eye. Such adaptations on the part of the organism do not necessarily imply any demonstrable change in its morphology or cultural reactions.

DISCUSSION.

DR. G. E. DE SCHWEINITZ, Philadelphia: I desire first to express my high appreciation of the paper which has been read and of the work which has been done by its authors. We have reached the point when it may be said that most cases of iridocyclitis result from the action of microörganisms or are of septic origin; in other words, that iridocyclitis must be studied rather as a complication in a symptom complex than as an independent disease; that the source of micro-organismal activity resides frequently in focal infections, and that such focal infections are not merely the place of entrance of the bacteria, but they represent places where, as Rosenow has said, these bacteria acquire properties which give them a wide range of affinities for various structures; that the question of circulating bacterial toxins as opposed to the lodgment, in the affected tissue, of bacteria themselves is a question still open to discussion. That double infections may exist is well known, and the authors have recognized the very important

point that because a man may have syphilis and iritis, it does not necessarily follow that if we eradicate the syphilis the iritis will get well; there may be still left a focal infection which may be the active cause of the iritis. Focal infections are most frequent in the buccal mucous membrane, including the rhinopharynx, the teeth, and the tonsils, but that there are many distant points in which the focal infection may reside, for example, the urethra and the seminal vesicles.

It is hardly necessary to refer to the importance of an accurate search for these focal infections. Inspection of tissues is not enough, even when the inspection is made by an expert. For example, the teeth cannot be found guiltless until the *x*-ray has been employed. The focal infection having been found, its removal does not by any means end the treatment, and the iritis does not necessarily get well because this focal infection has been eliminated. In point of fact, removal of the tonsil, for example, even when it is distinctly septic, is sometimes followed by a violent exacerbation of the iridocyclitis, just as I believe occasionally an active increase in arthritic symptoms follows a tonsillectomy.

Time does not permit more than a brief reference to the much discussed relation of so-called autointoxication to iridocyclitis. I may be permitted to repeat, what I have said elsewhere, that one may speak intelligently of autointoxication only when poisons are formed by the tissues of the body itself—that is, within the metabolism—and are not introduced through septic bacterial infections, and, as we have no accurate knowledge of these toxins, it would seem wise to discontinue the term gastrointestinal autointoxication, although freely admitting that gastrointestinal intoxications of bacterial or parasitic origin are potent sources of infection, and there is no doubt that in this sense such gastrointestinal intoxications, furnishing as they may bacteria or their toxic products, have a right to be included among the etiologic factors of iridocyclitis. Every clinician must certainly admit the enormous importance of regulating putrefactive processes in the intestinal tract in the treatment of iritis.

In this connection I would like to quote some sentences from a study on "The Action of Toxins and Protein Degeneration Products on the Eye," made by Dr. Alan C. Woods in the Laboratory of the Department of Research Medicine

of the University of Pennsylvania. We all know of the studies of Guillery on "Uveagifte." This name is unfortunately chosen, however, because the product produces general symptoms and many lesions throughout the body. The chief source of the poison is probably from the body of the bacteria itself, where it is formed by proteolytic action of the bacterial ferments. The poison, according to Woods, seems to be a high degeneration product of protein. It conforms to the class of ptomains, basic nitrogenous compounds closely allied to amino-acids. The action and origin of this poison offer possibilities for experimental confirmation of the theory of autointoxication in endogenous uveitis. Woods' final conclusions are as follows:

1. A poison can be obtained from cultures of B. prodigiosus, and probably from similar ferment-producing organisms.
2. This poison is not due to ferment activity, but is due to a degeneration product formed on the bacterial body and on the protein of the culture-media by the proteolytic action of the bacterial ferments. The poison comes under the class of ptomains.
3. Intravenous injection of this poison gives clinical symptoms and histologic lesions of a chronic infiltrative uveitis.
4. The poison is generally toxic to the animal, producing uneasiness, slight dyspnea, diarrhea, and prostration.
5. The ocular reactions observed following the intravenous injection of the poison are directly proportional to the general symptoms produced in the animal.

DR. WALTER L. PYLE, Philadelphia: Illustrative of the difficulties in determining the etiology of chronic iritis, with the usual involvement of the anterior uveal tract, I desire to report briefly the details of an interesting case at present under my observation.

In February, 1913, I was consulted by a civil engineer, aged twenty-five, a healthy, well-nourished man, weighing over 200 pounds. He had been suffering for a year from severe iridocyclitis, unrelieved by any treatment. The affection was first noticed in January, 1912, after exposure to intense cold and snow-glare while working on a survey in the Adirondacks. The left eye was first diseased, and five

months later the right eye became involved. The patient had been using aspirin practically continuously for one year to subdue the intense pain; and had also instilled in each eye for the same period a 1 per cent. solution of atropin, three times daily.

Although there were no clinical history or external signs of specific infection, a Wassermann test was made with positive findings in a noted medical institute in New York City. However, in this connection it must be noted that the specimen was sent by express from central New York State. Immediately after the positive blood-test, two doses of salvarsan were administered intravenously, and rigorous mixed treatment was instituted.

When I first examined the patient, there were all the signs of intense anterior uveitis in both eyes. The media were too cloudy to allow ophthalmoscopic examination. Inasmuch as there had been no internal medication for several weeks, and the anti-specific treatment at home and at a western springs resort had not produced any relief, I doubted that the inflammation was of syphilitic origin. Independently, I started a series of blood-tests in competent local hands; and through a period of nine months we were not able to secure any but undoubted negative findings.

The skull was carefully radiographed, and no significant sinus, intranasal, alveolar, or tonsillar disease was discovered. Careful physical and chemical examinations were also discouragingly negative. The tuberculin test gave a strong positive reaction, and I immediately began the routine tuberculin treatment.

In December, 1913, a sea voyage being agreed on, I advised the patient to consult Dr. Darier of Paris. Careful blood-examinations were made by him with negative results, but always prompt response to the tuberculin tests. The patient stayed with Dr. Darier for several weeks and had the advantage of the most modern local and general treatment for intraocular tuberculosis.

I saw the patient again late in the spring of 1914, and found the local inflammation so much improved in the left eye, the one first affected, that with proper optical correction V.= 6/30 +.

Early in 1915, through some misunderstanding in the

treatment at his home in central New York, there was a marked relapse of all the symptoms. In March, I found the right eye highly inflamed, sightless, and soft, with complete iritic synechiæ. Although the left pupil was partly dilated, this eye was also practically blind. I immediately ordered injections of vaccine of non-virulent T. B. (500,000,000 to c.c.), and staphylococci albus and aureus (600,000,000 to c.c.). The ordinary local treatment was maintained rigorously. In November, 1915, I prescribed thyroid extract twice daily, and also local light- and electrotherapy.

Soon after returning home the inflammation subsided and when I saw the patient one month ago both eyes were quiet and all pain had gone. He had not found it necessary to use aspirin for several months.

At the present time there is complete pupillary synechiæ in the right eye, with an irregularly dilated pupil. There is now well-developed double cataract, preventing any vision other than doubtful movement of the fingers. However, light-perception and projection were fair throughout the whole visual fields. The tension of the eyeballs was satisfactory. The patient is using the tuberculin treatment, and I hope later to perform a preliminary iridectomy, and ultimately extract the left cataractous lens.

The unfortunate aspect of this case is the rigid pursuance of exclusive syphilitic treatment after the positive Wassermann reaction. It must be remembered that in 1913, even in reliable hands, there was much chance for error in the newer blood-tests, particularly when the specimens were sent from a distance. I believe that this patient either never had syphilis or he had been cured by the salvarsan-injections; but, in any event, syphilis was not the cause of his unfortunate ocular condition, and fully a year had elapsed before proper treatment was instituted.

Dr. W. B. Marple, New York: At a recent meeting of the Academy of Medicine in New York an excellent paper on dental infections in relation to nasal and eye inflammations was read, and naturally the essayist was of the opinion that such infections played a very important rôle in ocular inflammations. He presented a patient suffering from iritis whom he had seen for the first time that day. Having

discovered also an apical abscess on that side of the face, he presented the case as one of iritis caused by dental infection! In my discussion of the paper I said that I could not understand or see how any one, by the appearance of the iritis, could say that the latter came from an apical abscess. There are no peculiarities or marks about an iritis from an apical abscess that distinguish it from an iritis from certain other causes, and this fact was demonstrated in my own service at the Infirmary recently in a case of iritis which was put upon the usual treatment immediately and in which patient we subsequently found an apical abscess. The patient began to improve immediately under the usual treatment, and notwithstanding the discovery of the apical abscess, I refrained from any treatment of this condition. The iritis was soon well, and it was evident that the apical abscess in this case had nothing to do with the iritis. This simply illustrates the importance of being sure that the suspected etiologic factor is the real one, and of not forgetting what the essayists and the President have said, that there may be a combined infection in many of the cases.

We may expect to read in our literature for some time to come reports of so-called ocular inflammations dependent upon dental infections. In other words, such infections will have a "boom" for a while in our literature. But, as Dr. Herman Knapp once said, "we cannot reach a satisfactory conclusion upon any subject until its 'boom' has subsided." Then we will probably realize that such infections do play a rôle, but not nearly so important a rôle as seemed to be the case during the "boom."

Dr. John E. Weeks, New York: I think that the light is beginning to shine on the cases of iritis that have passed heretofore as syphilitic; certainly such work as this is the work that will give us the proper information. We know that the text-books at the present time attribute syphilis as the cause of 65 to 75 per cent. of all the cases of iritis. It is written in our text-books that syphilitic iritis may resemble all other forms of iritis—that there may not be anything distinctive in the condition of the iritis that would lead us to suspect syphilis as the cause.

I would like to ask the authors of the paper if, in the cases

where papillary elevations appeared in the minor zone of the iris (papillary iritis), any other cause than syphilis was discovered; also whether they have found any cases where tumors such as we find in tubercular iritis have been due to any other cause?

In regard to the mixed infections, I have recently seen two cases of marked tubercular iritis in patients with syphilis with 4 + Wassermann. These patients also had pulmonary tuberculosis, with marked systemic disturbances.

The information that these experiments afford apparently makes it obligatory for all of us who encounter cases of iritis of doubtful origin, cases of uveitis, etc., to call in the internist and the gentlemen who are specialists in other branches of medicine to aid us in making a diagnosis and aid us in the proper therapeutic measures to be undertaken in the treatment.

Dr. Theobald: I would like to ask how Dr. Weeks determined, in those cases of 4 + Wassermann, that the iritis was due to tuberculosis.

Dr. Weeks: In the cases referred to the lesions in the iris were those we have learned to recognize as typical tubercular lesions, small elevations of a yellowish, grayish character, occurring in both zones of the iris. I do not think any one has found papillary syphilitic iritis in which the lesions are outside the minor zone of the iris. In tubercular syphilitic iritis, as we know, there are seldom more than two and almost always but one lesion. In one of the cases the effect of the tuberculosis on the scleral tissue was such that the intraocular pressure pushed the whole cornea forward, elongating the eyeball from before backward, a condition that we do not find in syphilis.

Dr. H. H. Briggs, Asheville, N. C.: It seems to me that 8 per cent., as shown on the chart as being tubercular, is rather excessive, and if you leave out the 17 cases of combined infection, you have practically 10 per cent. of tubercular cases. When you consider that most of the cases were in private practice and only a few in institutional work, the percentage of tubercular cases is more surprising. While I would not question the accuracy of the statistics, I do think

that in making our diagnosis we often give undue importance to the fact that the patient has a general tuberculosis or responds to the tuberculin test. This test, so far as ocular tuberculosis is concerned, is not so important as is the Wassermann test in syphilitic lesions of the fundus. I do not think that we ordinarily find 8 or 10 per cent. of tuberculous cases.

DR. M. L. FOSTER, New Rochelle, N. Y.: I would like to report a case under the head of "no cause found." The patient, a grandmother, was very healthy and had been healthy all her life. She said she had never had a sickness and never had had a pain, except those incident to childbearing, until the occasion on which she called me in, and she was then over sixty years of age. She presented absolutely no symptoms or history of syphilis or tuberculosis. I did not inquire regarding gonorrhea. So far as the dental and tonsillar conditions were concerned, I did not investigate carefully, but she told me she had never had toothache or sore throat, and had never had, as she said, a pain in her body. She denied ever having had even ordinary muscular rheumatism, from which most of us have suffered more or less. It is customary now to omit rheumatism from the list of causes of iritis, but in the investigation I had the urine examined and found a great overproduction of uric acid. She was put on treatment for rheumatism and in a short time afterward this obstinate iritis proceeded to get well. She was kept on the treatment for several months after the iritis was well, until the amount of uric acid in the urine was approximately near normal. I have had her under observation for some three or four years since and she has never had another attack. What would you say that was unless it was a real, old-fashioned, rheumatic iritis?

DR. SAMUEL THEOBALD, Baltimore, Md.: I wish to say in answer to the question asked by Dr. Weeks in regard to the ground for diagnosis in connection with the Wassermann, that I have occasionally seen just such cases. One I have in mind, in which there were numerous small nodules on the iris, which I am quite sure were not confined to the pupillary zone, was shown at the Baltimore Eye, Ear, and Throat Hospital as an interesting example of tuberculous iritis. The

picture being strongly suggestive of condylomatous iritis, I naturally asked if a Wassermann had been made, and was told that it was in process, and had not yet been reported upon. The report later showed positive reaction, and the case was then put on anti-syphilitic treatment and the suspected tuberculous iritis subsided.

DR. BROWN: In reply to Dr. Weeks' question as to our grounds for diagnosis of tuberculous cases, we did not encounter any cases with tubercular nodes in the iris. Our diagnosis was made upon the occurrence of an iritis in patients with active tuberculosis elsewhere and without any other adequate cause. Like Dr. Marple, we have had cases with a 4 + Wassermann, and yet in such combination with clear-cut tuberculosis, dental and tonsillar infection that we had to put them in the combined group, for we could not determine which factor was the real causative one.

DR. IRONS: In answer to Dr. Briggs' question as to the use of tuberculin in diagnosis, we have not made a diagnosis of tuberculous iritis on the tuberculin test alone, any more than one would make a diagnosis of syphilitic iritis on the Wassermann alone, or a diagnosis of gonococcal iritis on a positive complement-fixation test. In the instances of iritis which we believed to be tuberculous, three were in clinical patients and five were in private patients. These were all very carefully studied to discover any other infections, but in six of the eight patients there were no other coincident infections found. There is, of course, no reason why a person suffering from tuberculous iritis may not also have chronic tonsillitis, apical abscess, syphilis, or something else, but it so happened that in these cases other infections were not present.

One other point. The more we have studied these cases of iritis the more impressed we have been with the necessity of repeated, painstaking examinations and reviews of the data obtained before drawing final conclusions as to the etiologic diagnosis. Perhaps three-fourths of these patients have been studied over periods of from six to eight months, the studies continuing after we have made a provisional etiologic diagnosis, and such subsequent studies have occasionally caused us to revise our first opinion.

A CASE OF BLEPHAROCHALASIS—A VARIETY OF DERMATOLYSIS.

ROBERT L. RANDOLPH, M.D.,
Baltimore, Md.

Alice Price, aged fifteen, has had since birth an unusual condition of the upper lids, consisting in considerable looseness of the skin and thinness of the connective tissue in this situation. One can also notice lichen-like changes in the lids, which no doubt account for the periodic attacks of itching which occur from time to time and which provoke a great deal of scratching and consequent swelling of the lids. For weeks at a time, however, the relaxation of the skin is the only trouble present. This relaxation, atony, or dermatolysis, or, as Fuchs suggests, blepharochalasis, is a very rare disease, and only a few cases have found their way into literature. According to Fuchs,* the trouble is always bilateral. The skin, owing to its loss of elasticity and thinning, is thrown into countless folds criss-cross in all directions, such as one sees in advanced senile atrophy and relaxation of the skin, and, as Fuchs describes it, presenting a tissue-paper-like appearance. Very frequently there are, at the most convex point on the lids, numerous small dilated veins, such as one sees in elderly persons with red cheeks. There is no hypersensitiveness of the skin. The changes, as will be seen, are pronounced between the eyebrow and the upper edge of the lid. In conclusion, it should be said that the disease is one of early childhood, starting at puberty. It must be said, however, that, so far as I can learn from the

* Wiener klinischer Wochenschrift, No. 7, February, 1896.

Blepharochalasis. Much magnified picture. Shows characteristic tissue-paper-like wrinkling in the skin of the upper lids.

mother, this child has had it from earliest childhood. The accompanying photograph show well the condition described. Two cases are reported by Weidler* and two by Stieren.†

DISCUSSION.

DR. EDWARD B. HECKEL, Pittsburgh, Pa.: Dr. Randolph's patient apparently presents the condition usually described as blepharochalasis, and in this case is bilateral and shows no ptosis. I have under observation a girl of fifteen, with a typical so-called blepharochalasis involving one eye only, and with a well-marked complete ptosis, which raises the question whether or not ptosis is not always a concomitant condition in monolateral cases and always absent in bilateral cases. In my case the condition followed repeated attacks of swelling of the lid, very likely an angioneurotic edema.

DR. EDWARD STIEREN, Pittsburgh, Pa.: This in my opinion is a typical case of blepharochalasis, having the lax, redundant integument with the wrinkled cigaret paper appearance of Fuchs. By palpating under the upper rim of the orbit rounded bodies can be felt, which no doubt is orbital fat coming forward. When the patient leans forward, the fulness of the upper lids is much increased—a diagnostic point to which I referred in my article in the Transactions of this Society for 1914. An interesting feature of Dr. Randolph's case is that the condition appears to be congenital; whereas in the majority of reported cases it occurred early in puberty.

* Trans. Amer. Med. Assoc., Section Ophthalmology, 1913.

† Trans. Amer. Ophth. Soc., 1914.

OBSERVATIONS UPON HERPES CORNEÆ "FEBRILIS," WITH REFERENCE, ESPECIALLY, TO ETIOLOGY.

SAMUEL THEOBALD, M.D.,
Baltimore, Md.

Unless my experience has been exceptional, herpetic keratitis is a more common affection than it is usually supposed to be. A habit with me, for many years, of testing the sensibility of the cornea in every case of superficial keratitis in which the etiology was in doubt may, perhaps, account in some measure for this conviction; for unquestionably the most characteristic sign of herpetic keratitis is corneal hypesthesia.*

It may be well to state at the outset that the term "herpetic keratitis" is used, as Fuchs employed it, in a broad sense, neuropathic keratitis, the post-malarial keratitis of Kipp, and the dendritic keratitis of Emmert being regarded simply as types of corneal herpes, the essential and common feature of these different types being a disturbance of the nerve supply of the cornea.

It does not seem that the qualifying term "febrilis," commonly applied to simple herpes of the cornea, is well chosen; for, though herpes labialis, and perhaps more general facial herpes, not infrequently occurs as an accompaniment of "colds," attended by fever, this, according to my observation, is not true of corneal herpes, which, if we except the post-malarial cases, more often than not occurs without assignable cause.

* As the sensibility of the cornea differs greatly in normal eyes, one should be careful, in determining the existence of hypesthesia, to make the test upon both eyes, as a matter of control. A spill of tissue paper I have found convenient for this purpose.

Beside the corneal hypesthesia, which is very rarely confined to the infiltrated or ulcerated areas of the cornea, as some maintain, the characteristic signs and symptoms of herpetic keratitis, commonly accepted, are the unilateral character of the affection; the diffuse and irregular form of the corneal opacities, sometimes of the dendritic type, but more often presenting a "map-like" appearance; the persistence of the opacities notwithstanding the fact that the substantia propria is seldom deeply involved; the obstinacy of the affection, and, not infrequently, a decided tendency to recurrence. The severe pain, which some describe, is not, in my experience, a usual symptom.

I have long felt that, in all probability, the primary lesion which gives rise to simple herpetic keratitis, and which explains the corneal hypesthesia, is located in the ciliary ganglion. This lesion, it may be fairly assumed, is comparable to that which occurs in the Gasserian ganglion in herpes zoster ophthalmicus; but, since it involves only the ciliary nerves, the resultant disturbance of nutrition, the hypesthesia, and the inflammatory changes which follow are necessarily confined to the eye itself, differing radically in this respect from those which are usually observed in herpes zoster ophthalmicus, where the primary lesion is in the Gasserian ganglion, and not only the eye, but the lids, the forehead, and scalp, and exceptionally the side of the nose, are involved.

I am aware that this belief is not in accord with the commonly accepted view regarding the character of the ciliary ganglion, which holds that it is purely an efferent ganglion, and that the fibers from its sensory root are not in relation with cells in the ganglion; in other words, that the ganglion is not a "station" for these sensory fibers.

If this view were correct, it is manifest that a lesion confined to the ciliary ganglion could not cause the corneal hypesthesia and the accompanying nutritional and inflam-

matory changes which are characteristic of simple herpetic keratitis. But it may be safely asserted that the correctness of this conception of the nature of the ciliary ganglion has not been definitely demonstrated.

In his paper, "Ueber die Doppelnatur des Ganglion ciliare,"* published, it is true, in 1881, W. Krause, as the result of research made upon rabbits, reached the conclusion that the ciliary ganglion was not a simple sympathetic ganglion, but was double in its nature, one portion of it resembling the ganglia upon the posterior roots of the spinal nerves.

More recently Frederick Walton Carpenter, of the Harvard Laboratory of Comparative Zoölogy, as a result of his exhaustive study of "The Development of the Oculomotor Nerve, the Ciliary Ganglion, and the Abducent Nerve in the Chick,"† reaches a similar conclusion, at least as to the double nature of the ciliary ganglion. "The evidence," he states (p. 205), "derived from the study of both its development and its histology, points to the double nature of the ciliary ganglion of the fowl," and again, on p. 207, he says: "The larger ventral region of the ciliary ganglion bears little resemblance to a sympathetic ganglion. The cells in it are of greater size, pericellular fibrils are not abundant, and its neuraxons are heavily medullated." He differs, however, from Krause, whom he quotes as looking "upon the [ciliary] ganglion in vertebrates lower than mammals as a homologue of a spinal ganglion," in that he considers the ciliary ganglion of the fowl "as composed of cells which fall into two categories, one being, as far as the evidence goes, in all essential respects typically sympathetic, the other belonging to neither the sympathetic nor cerebrospinal system." On p. 144 he says: "It," the ciliary ganglion, "is almost invariably stated to be sympathetic in nature (as first suggested by Arnold), although it differs from typical sympathetic ganglia

* Morphol. Jahrb., viii, p. 43.

† Bulletin Museum of Comparative Zoölogy, January, 1906, xlviii, No. 2.

in giving origin to medullated peripheral nerves (the short ciliaries) instead of non-medullated fibers," and on p. 150 he alludes to the investigations of Stefani, who "was led to the conclusion that the short ciliary nerves have their centers, *i. e.*, their ganglion-cells, in the ciliary ganglion, by observing the effect on the cells of that ganglion when atropin is applied to the eye."

It may be added, as not without interest, that Fuchs, whose high authority and exact knowledge we all recognize, evidently did not accept Arnold's views as to the purely sympathetic character of the ciliary ganglion, else he would not have made the statement: "An inflammatory affection of the trigeminus, which is located either in the trunk of the nerve itself or in the Gasserian or *ciliary ganglion*, lies at the bottom of herpes zoster ophthalmicus."*

It would seem, then, that there is no well-founded anatomic or physiologic objection to my belief that the primary lesion responsible for simple herpetic keratitis is located in the ciliary ganglion.

As opposed to the view that the lesion may be in the Gasserian ganglion, it may be said that, while simple herpes of the cornea, as has been stated before, is of relatively frequent occurrence, herpes zoster ophthalmicus, which, as commonly observed, we know *is* due to disease of the Gasserian ganglion, is a rare affection; and this contrast is emphasized by the fact that the picture which the latter presents is so typical that an error in diagnosis can scarcely be made, even by a tyro, while it is not improbable that a considerable number of cases of simple herpetic keratitis are not recognized as such, even by those who cannot be regarded as novices. Furthermore, if the primary lesion were in the Gasserian ganglion, it is difficult to explain why the peripheral manifestation should be confined to the cornea,

* Text-book of Ophthalmology, Amer. Edit., p. 621. I have taken the liberty of italicizing the words "ciliary ganglion" in this quotation.

and why this peculiar sort of lesion should occur so much more frequently than the more extensive pathologic condition which gives rise to the usual type of herpes zoster ophthalmicus.

The only other explanation of the etiology of simple herpetic keratitis would seem to be that the ciliary nerves, in their short course from the ganglion to the posterior pole of the eyeball, are primarily involved, which, to say the least, does not appear to be a probable hypothesis.

As Dr. W. H. Howell, head of the physiologic department, and Dr. Florence R. Sabin, of the anatomic department, of the Johns Hopkins Medical School,—both of whom have afforded me helpful assistance in the preparation of this paper,—feel that by the employment of modern methods of research it should be possible to clear up the present uncertainty regarding the true character of the ciliary ganglion, I am not without hope, if the right man can be found to undertake the exact and painstaking work required, that under the supervision of one or the other this much to be desired end may be attained.

As to the treatment of herpetic keratitis, I may say that the local remedies which I have found most useful are holocain and atropin, and that not infrequently one, sometimes both, of these are supplemented by instillations of dionin.

Holocain is usually my first choice, to avoid the inconvenience of mydriasis, and it is employed in a one-grain—if there is much ciliary irritation in a two-grain—to the ounce solution, with the addition always of boric acid, ten grains to the ounce. If the response to this is not satisfactory, atropin two to four grains to the ounce, with the addition of boric acid in the strength just mentioned, is substituted. Not infrequently, as has been indicated, both of these agents are employed, and if the keratitis proves intractable, dionin, in 5 per cent. solution, applied twice or thrice daily, in conjunction with one or the other, possibly with both, is tried.

The constitutional remedies which I have learned to place confidence in are quinin, in generous doses, and potassium iodid, each of which is given with the idea that I am attacking the primary lesion supposedly in the ciliary ganglion. The former, commonly first prescribed, is given in doses sufficient to produce some cinchonism—usually three grains four times a day. If the response to this, after a fair trial, is not satisfactory, potassium iodid in moderate doses, usually ten grains three times a day, is given instead. Arsenic, which some think useful, I have not found as efficacious as the two drugs mentioned.

A rapid subsidence of the keratitis is seldom observed, and one should be prepared and, what is more important, should prepare the patient for a somewhat tedious recovery. The first observed evidences of improvement, as is generally recognized, are a diminution of the ciliary irritation—the photophobia, lacrimation, and blepharospasm—and a lessening of the conjunctival injection, which is only exceptionally very pronounced at any stage, to be followed later by a gradual change for the better in the condition of the cornea, the restoration of lost tissue, which is seldom very considerable, the reformation of the epithelium, and, last of all, the disappearance of the opacities, which, not very infrequently, is incomplete, and of the hypesthesia, which is commonly long delayed and which, I am inclined to believe, is also, in some instances, incomplete.

DISCUSSION.

DR. F. H. VERHOEFF, Boston, Mass.: In regard to the nomenclature of these cases, I do not quite agree with Dr. Theobald's plan of calling them all herpetic. It seems to me better to classify them all as neuropathic keratitis, and specify certain forms by the terms herpetic type, dendritic type, etc., for they are all neuropathic in origin. Neuroparalytic keratitis is simply a severe neuropathic keratitis. In the textbooks the nomenclature is greatly confused.

In regard to the malarial cases, most of them are dendritic in type. In a great many the patient has had malaria years ago and there is no evidence of it at the time of the attack of keratitis. It seems to me that the explanation of this fact is that when the patient had malaria the Gasserian ganglion was affected and thus rendered more susceptible to the more common causes of neuropathic keratitis.

The question of whether in cases of neuropathic keratitis it is the Gasserian or ciliary ganglion that is at fault, I do not think is definitely settled. It seems to me, however, that usually it must be the Gasserian. We know that in herpes zoster the Gasserian ganglion is affected, as shown by microscopic examinations, and we also know that about every known form of neuropathic keratitis may occur in this affection. It is, therefore, reasonable to suppose that when the same forms of keratitis occur under other conditions they likewise must be due to lesions of this ganglion. Moreover, in many cases of neuropathic keratitis there is herpes of the face, and this, of course, could not be caused by a lesion of the ciliary ganglion.

In superficial punctate keratitis, which is a special form of neuropathic keratitis, the corneal spots are mostly central, and there is seldom a spot nearer than a millimeter to the limbus. This sort of clinical evidence goes to show that the ciliary ganglion may be the one at fault here, but I am more doubtful of this than I was at the time I published a paper on this subject. A possibility that suggests itself also is that in herpes zoster the ciliary ganglion may be secondarily involved. As a matter of fact, we know that in certain cases of zoster there may be paralysis of some of the ocular muscles, indicating extension of the inflammatory process from the Gasserian ganglion to the affected motor nerves, and in one case I observed the occurrence of an Argyll-Robertson pupil on the affected side, indicating extension to the ciliary ganglion. Then, too, the corneal involvement does not usually take place at the same time as the skin eruption and very often not until the latter is entirely healed. On the whole, however, I am inclined to believe that the Gasserian ganglion is alone at fault in the vast majority of cases of neuropathic keratitis.

DR. THEOBALD: I wish only to say that the nomenclature

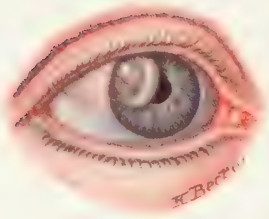


Fig. 1.—Daylight view of opacity.

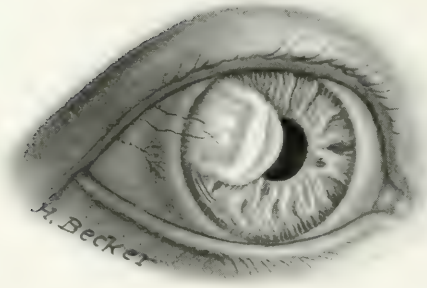


Fig. 2.—The opacity seen under oblique illumination and magnification.

which I followed, and which has invited criticism, was, as stated in the paper, that used by Fuchs; and to say, further, that, in my experience, herpetic keratitis is only exceptionally accompanied by facial herpes.

TWO PATIENTS WITH UNUSUAL CORNEAL OPACITIES.

JAMES BORDLEY, JR., M.D.,
Baltimore, Md.

While the corneal opacities, illustrations of which I show, may not be unique, they are certainly rare. I have not made an exhaustive study of the literature, but after a rather careful search have been unable to discover any case identical in character. I might add that, in a personal clinical experience covering a fairly large variety of corneal lesions, I have never observed such an opacity except in the two cases which I wish to describe.

In both of these patients the opacities were alike and the causative agent was the same. Both patients were injured on shipboard, one by walking into the edge of a low deck, the other by a splinter which was broken off a chicken coop behind which the patient was seeking protection from shell-fire from one of the German commerce raiders off the South American coast.

In both cases the traumatism was through the closed lids. The opacity, starting from the outer corneal margin, slowly advanced toward the center. In one of the patients the cloud extended across the pupillary area; in the other the extension was not quite to the median line of the cornea. In neither case was the epithelium elevated or otherwise disturbed.

In one case the opacity was watched from its incipiency.

Starting at the corneal limbus as a small, densely white mass about the size of an ordinary pin-head, an advancing head gradually developed. This head was at its pupillary border a translucent cloud, and just behind the cloud was a densely white, opaque, arch-like formation with the bases of the arch resting on the corneal limbus. Immediately beyond this arch was an almost transparent, cloudy area. As the advancing head moved toward the center the vertical diameter of the opacity increased.

The sclerotic was in no wise disturbed nor was the iris. A few of the conjunctival vessels were dilated in the late stages, but primarily the only structure affected was the cornea.

The opacity continued to expand for about fourteen months, then the process became retrogressive. The opacity cleared from the periphery toward the center. The only abnormality which now remains, twenty-three months after the initial change, is a very small, apparently pigmented, opacity which looks not unlike a chalky deposit.

The other patient was seen only after the opacity had completely covered the pupil. In appearance it was identically the same as in the first patient. Unfortunately, this patient was called to Europe before there was any evidence of retrogression of the opacity, consequently the outcome I am unable to record. As can be readily imagined, I had an opportunity, as a result of the long duration of the disease, to almost exhaust the pharmacopeia of "remedies." I must candidly say, however, that if medicines played any part in the recovery of the first patient, the only one treated from start to finish, I was unable to recognize that fact. Dionin was used every day for over a year, and possibly it influenced the progress of the disease.

The illustration (Fig. 1) shows a daylight view of the eye. This picture was drawn at a time when there was dilatation of the conjunctival vessels following the instillation of dionin.

Under usual circumstances only three conjunctival vessels were dilated.

Fig. 2 was made under magnification and oblique illumination. This illustrates the formation of the opacity most beautifully. Mr. Becker, who made the drawing, was much disturbed in making this last picture by the constant change in breadth and density of the opaque portion of the opacity. He made drawings frequently for three weeks, and in every one the arch had a somewhat different form.

OPHTHALMOSCOPIC WORK IN THE BRITISH BASE HOSPITALS.

ALLEN GREENWOOD, M.D.,
Boston, Mass.

Last summer the writer had the privilege of working with the Harvard Surgical Unit in the British Expeditionary Force Base Hospital No. 22, in France, for three months, and during that time a great many interesting fundus changes were observed and watched from day to day and often seen several times a day.

The Fundus in Trench Nephritis.—A good many soldiers are sent back from the trenches suffering from what has been termed "trench nephritis." There has been more or less controversy as to the cause of this, and at one time the urine in all these cases was tested to see if it might be due to drinking water poisoned with bichlorid of mercury. It has seemed to the writer that a satisfactory explanation is that this form of acute nephritis results from a lessening of the skin elimination produced by standing for days and days in water-soaked clothing, and often standing in water up to the waist.

These men come into the hospital with a very marked

general edema, which in most cases is so extensive that pitting can be produced on the back of the fingers between the joints. The edema is also especially marked in the face. The urine, which is scanty, shows cloudy and at times slightly smoky. Examination of the urine shows a large amount of albumin, a few hyaline and granular casts, with some renal cells and occasional blood-cells. The most remarkable feature shown by these cases was the extremely rapid subsidence of all the symptoms soon after the men had been placed in a warm, comfortable bed.

The British War Office requires the filling out and sending to the Boulogne station of a special blank, giving the results of urinary examination in all these cases. This blank also requires that a fundus examination be made. In the earlier cases of "trench nephritis" that came to Hospital 22 this fundus examination was not made until after the practical subsidence of the general edema, and no changes were found.

It seemed, however, to the writer that there must be some disturbance with the optic nerve and retina where there was such a great amount of general edema, and it was determined to make examinations as soon as the patients entered the wards. Accordingly, at 2 o'clock one morning, when six cases came into Ward A5, a fundus examination was made before the patients were put to bed, with the following results:

D. P., neuroretinal edema.
A. D., neuroretinal edema.
S. D., optic neuritis.
B. W., optic neuritis.
A. O., slight edema of discs.
W. D., fundi normal.

The five cases showing fundus changes presented marked general edema, with puffing of the face and eyelids. The sixth case had very little edema, but the urinary examination was in keeping with the others.

These patients were all seen again after six and a half hours' rest in bed and profound slumber, and all showed a marked decrease in the fundus changes.

Twelve hours later, in the first two cases, the fundi looked normal, the next two showed a slight neuritis, the fifth would ordinarily have passed for a normal fundus, the remains of the edema being so slight, while the sixth still showed normal.

Another twelve hours later, after a full night's sleep, there was no sign of any fundus disturbance in any of these cases.

Subsequent cases of "trench nephritis" showed similar fundus changes if seen immediately after admission. In no case was there the slightest evidence of any degenerative processes, the whole picture being that of an acute nephritis of sudden onset and equally rapid recovery. It will be interesting to note if in the future any of these cases show signs of impaired kidney function.

The Fundus in Brain Injuries.—As might be expected, practically all perforating brain injuries resulted in a more or less extensive meningitis, which was evidenced in the examination of the fundus by an optic neuritis, and, where the cranial drainage was poor, the resulting increased cerebral pressure was evidenced by a choking of the disc.

Most of the cases of perforating and furrow skull injuries at the Twenty-second General Hospital, of course, came to operation for the removal of depressed inner plates and splinters of bone and the institution of suitable drainage. These cases had repeated fundus examinations, and at first the ophthalmoscope revealed nothing, but on succeeding days in all cases which developed symptoms of meningitis there was noted a beginning optic neuritis. Sometimes the optic neuritis would be discovered before marked meningeal symptoms had developed. Where there were evidences of increased cerebral pressure there appeared a rapid choking of the disc, which would subside as rapidly on the renewal of adequate drainage. So clearly defined were these changes

that the surgeons at the hospital came to depend on the frequent reports of fundus examination for one of their guides as to the necessity for reopening the cranial wounds for reëstablishing the proper drainage. The following cases in Ward B6 are illustrative:

A. D. G., entered the ward September 27th with a gunshot wound of the right side of the head, point of entrance being about an inch above the right eye, point of exit in the upper part of the right occipital region. Both wounds were the same size, having been made by a high velocity, sharp-pointed, steel-jacketed bullet. A double trephining was done, drainage passed through the brain, and on the fifth day he developed an optic neuritis and other signs of pressure. The reëstablishment of better drainage immediately lessened the amount of neuritis, and this had to be repeated several times before the optic neuritis finally subsided.

H. F. Gunshot wound of right frontal region, with prolapse of brain substance. On the fifth day after operation there appeared a slight neuritis and signs of compression, which were quickly and permanently relieved by improving the drainage.

H. T. Furrow wound of left parietal region of the skull with depressed inner table and exposure of the brain. This patient also developed an optic neuritis on the fifth day after operation, and in this case there was also immediate relief upon bettering the drainage.

H. G. Gunshot furrow wound of the left occipital region with depression of the inner table, causing a right homonymous hemianopsia. This case progressed favorably and without any development of optic neuritis.

P. H., W. A., and W. L. All had furrow wounds of the skull, with depression of the inner table, necessitating operative interference, but in none of these cases did any optic neuritis develop as a result of meningitis or compression.

The foregoing cases all came into one ward from the same convoy. In other wards of the hospital there were a number

of similar cases where the fluctuation in the amount of optic neuritis and choking of the discs were looked upon as indications for or against renewed attempts to establish proper intracranial drainage.

DISCUSSION.

DR. H. F. HANSELL, Philadelphia, Pa.: I would like to ask whether the optic neuritis was always double, and if so, was it more marked on the injured side?

DR. GREENWOOD: I mentioned these cases briefly because of their being fundus cases. The optic neuritis was practically always double, and the greater elevation of the disc was on the injured side.

DR. S. D. RISLEY, Philadelphia, Pa.: I would like to inquire whether the general edema subsided with the edema of the fundus?

DR. GREENWOOD: Inside of six or eight hours the edema had largely disappeared; in twenty-four hours there was no sign of edema except perhaps a little pitting on the skin. When these men come into the hospital you cannot imagine the dragged-out appearance and the extreme prostration as the result of several months' suffering in the trenches, and twenty-four hours in bed did more for them than any other treatment. I am in hopes that at the meeting of the Society next year I can give a more detailed account of some of the eye work in the hospitals during the coming summer.

DR. W. E. LAMBERT, New York: Were any of these cases of trench nephritis complicated with the effects of poisonous gas?

DR. GREENWOOD: There was no complication with poison gases; evidently the poor skin elimination of the men was due largely to their water-soaked clothing.

REPORT OF THE REMOVAL OF A TUMOR AT THE APEX OF THE ORBIT WITH PRESERVATION OF THE EYEBALL IN A CASE OF PLEXIFORM NEUROMA OF THE EYELID. MICROSCOPIC EXAMINATION.

ARNOLD KNAPP, M.D.,
New York.

The patient's previous history is that, at the age of nine months, presumably after a fall, a swelling appeared in the right upper lid; she was operated upon for this swelling by different ophthalmic surgeons at the age of two and one-half, five, twelve, and fifteen years, respectively. At the beginning of 1912, when the patient first came under my observation, she was a normally developed though mentally backward girl of seventeen years, with a number of round, pigmented spots on the back and legs. The upper right lid was thickened, hung down, and could not be raised; there were irregular scars in the skin of the upper lid which extended to the temple. The eye was normal, proptosed, and there was a corneal scar. A tumor of the orbit was presupposed and an exploratory operation recommended.

Operation.—January 5, 1912. After throwing the outer bony wall of the orbit backward, as in the Krönlein operation, a horizontal incision was made in the periosteum above the level of the external rectus muscle. The index-finger was carefully inserted and the contents and walls of the orbit explored; a round, elongated tumor was felt at the apex of the orbit, just underneath the optic nerve. A path was carefully dissected in this direction, and with the aid of blunt retractors the tumor was exposed and found adherent to the sheath of the optic nerve. This adhesion was separated, a capsule was isolated and opened, and the tumor removed. The wound was closed with sutures after replacing the bone-flap. The eyelids were sutured together to

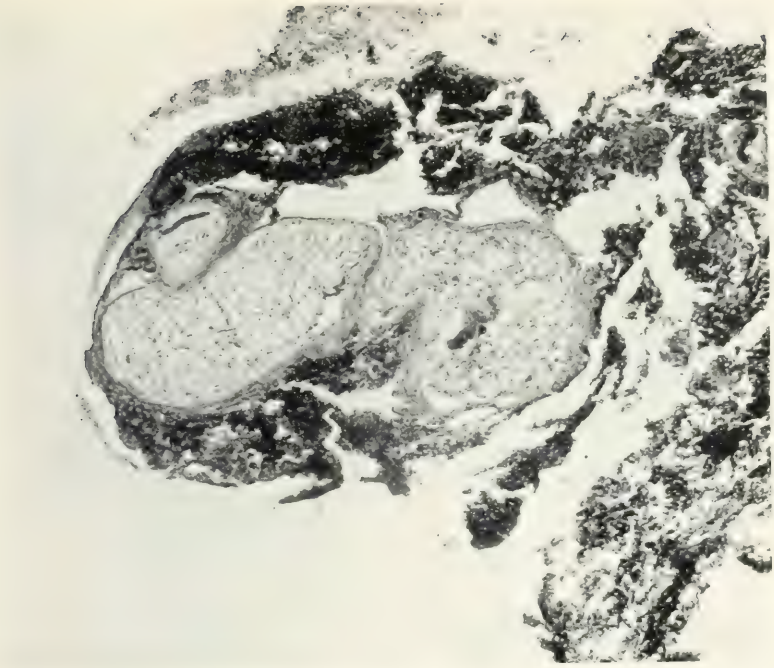


Fig. 1.—The tumor (*plexiform neuroma*) of the eyelid.

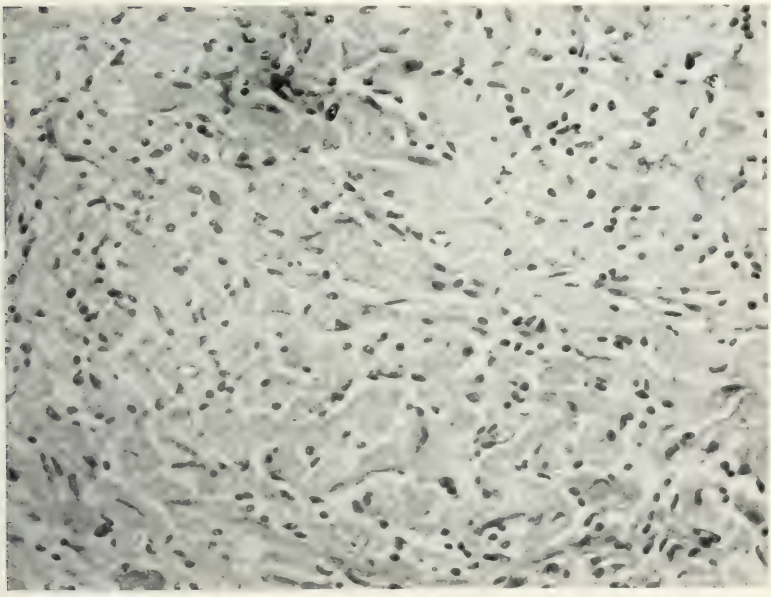


Fig. 2.—The tumor (*neurofibroma*) at the apex of the orbit.

prevent the chemosis and lagophthalmos which are apt to follow orbital exploratory operations. Recovery uneventful.

The tumor measured 22 x 12 mm. It was soft; the cut section looked like a myxomatous gland. Microscopically it showed the structure of a neurofibroma (Fig. 2); at the periphery normal and degenerated medullated nerve-fibers were present, beautifully shown in sections which Dr. F. H. Verhoeff was kind enough to stain with phosphotungstic acid hematoxylin.* As one of the previous operations had been performed at the old hospital, some of the tumor which was removed from the eyelid was available and new sections were stained by Dr. Verhoeff; these showed the centrally placed medullated nerve-fibers and the other characteristics of the main growth as it occurs in the eyelid (Fig. 1).

Plexiform neuroma is a well-defined though unusual tumor. Its frequent presence in the eyelid and adjoining temporal and frontal regions has brought it particularly to the attention of the oculist, and one of the earliest reports is by de Schweinitz† before this Society twenty-five years ago. Alexis Thomson‡ believes that plexiform neuroma must be included in the group of diffuse or generalized neurofibromatosis, for it is the result of a localized fibromatosis following the distribution of a particular nerve or a nerve-plexus. The two conditions may be met with in the same individual. He has collected 62 cases. Of 58 cases, 18 occurred in the temple, forehead, and upper eyelid, 14 in the posterior part of the neck and behind the auricle.

Michel§ states that the distribution of this tumor corresponds to the distribution of the first branch of the fifth nerve,

* Verhoeff's improved elastic tissue:
Stain with double quantity of ferric chlorid, twenty-four hours.
Rinse in water.
Mallory's phosphotungstic acid hematoxylin, twenty-four hours.
Rinse in water.
Alcohol, oil (if celloidin section), balsam.

† Trans. Amer. Ophth. Soc., 1891, vi, p. 48.

‡ Neuroma and Neurofibromatosis, Edinburgh, 1900.

§ Michel: Graefe-Saemisch, second edition.

though there is no pain, as a rule. Aubineau and Civel* describe plexiform neuroma as having a triad of symptoms, consisting of cutaneous tumors, tumors of nerves, and pigmented spots, with general functional disturbances and arrest of development. These three principal symptoms were all present in our patient. The main or cutaneous tumor was situated, as is usual, in the upper lid and in the adjoining temporal region. The pigment spots on the skin of the back and legs and the retarded mental development are not unusual symptoms.

In addition to thickening of the skin of the half of the face (elephantiasis) and involvement of the bones of the face, the orbital complications are of particular interest. The tumor sometimes extends into the orbit. Sachsalber† described a plexiform neuroma of the orbit with buphthalmos. The growth was made up of coils of enlarged and varicose nerves lying in a mass of fibrous tissue. The association with buphthalmos has been noted several times. In a case of Snell and Treacher Collins‡ the ciliary nerves were affected in their passage through the sclera. Rockliffe and Parsons§ reported on a case where the entire orbit was filled with a mass consisting of nerves thickened and tortuous from fibromatosis. Beard and Brown‖ described an orbital neuroma involving the supraorbital nerve; and Tertsch** observed a case of orbital tumor in which the lacrimal nerve was probably affected.

A complication of great interest is the tumors of the nerves. Thomson found, in 45 cases of generalized neurofibromatosis, the cranial nerves affected in about two-thirds, the vagus in 29, and the trigeminal in 12. According to

* Archiv. d'Opht., 1911, xxxi, p. 808.
† Deutschmann's Beiträge, 1898, xxvii.
‡ Trans. Ophth. Soc. Unit. Kingd., 1903.
§ Trans. Path. Soc., 1904.
‖ Archiv. of Ophth., 1906, vol. xxxv.
** Graefe's Archiv, 1903.

Marx,* tumors have been described in the third, fourth, and supraorbital branch of the fifth nerves. Fehr† reports on the case of a man with multiple skin tumors who showed a hemorrhagic neuroretinitis and a central scotoma in one eye. This developed into thrombosis of the central retinal vein, and a diagnosis of neurofibroma of the optic nerve was made principally by exclusion, though an exploratory orbital operation revealed nothing.

In Aubineau and Civel's case there was, in addition to the tumor of the upper eyelid, a paralysis of the external rectus of that side. These authors collected six cases of intracranial localization from the French literature alone.

The case which I had the opportunity of observing was remarkable for the presence of a neurofibroma at the apex of the orbit, in addition to a plexiform neuroma in the lid. It cannot be stated with certainty to what particular nerve this tumor at the apex of the orbit was related; it probably was the nasal branch of the ophthalmic nerve, as this is the only sensory nerve just below the optic nerve in this position. At operation it was found closely adherent to the optic nerve sheath.

DISCUSSION.

DR. E. C. ELLETT, Memphis, Tenn.: I would like to ask if Dr. Knapp did the Krönlein operation, and how old the patient was.

DR. ARNOLD KNAPP, New York: The patient was a girl, seventeen years of age, and the operation was the Krönlein operation. It is difficult to say to what nerve this neurofibroma was connected at the apex of the orbit. As it seemed to be situated just below the optic nerve, it may have been in relation to the nasal branch of the ophthalmic division of the fifth nerve.

* Zeitsch. f. Augenheilk., 1908, p. 528.

† Centralbl. f. prakt. Augenheilk., 1913, p. 233.

AN UNUSUALLY LARGE OSTEOMA OF THE FRONTAL, ETHMOIDAL, AND SPHENOIDAL SINUSES INVOLVING THE ORBIT AND ANTERIOR CEREBRAL FOSSA, PRESENTING NO SUBJECTIVE SYMPTOMS OTHER THAN PROPTOSIS OF THE EYEBALL.

C. A. VEASEY, M.D.,
Spokane, Washington.

Osteoma of the frontal sinus is sufficiently infrequent of itself to warrant the report of isolated cases, and the following history, presenting several unusual features, is herewith submitted:

Mrs. A. B., aged thirty, was brought to consult me on December 1, 1915, because of some proptosis of the right eyeball which was first observed eleven years before, and which had been increasing very slowly during that period. She stated that her father had taken her to consult some oculist in Kansas City seventeen years before consulting me, because of a so-called "enlargement of the right eyeball," but she herself was quite sure that the displacement, even though it might have been present at that time in a moderate degree, did not become markedly noticeable until about eleven years ago.

The patient's family history was negative. So far as she knew no member of her family had ever had a tumor of any kind. She had been married for several years, enjoyed comparatively good health, and, excepting an occasional dull aching pain in the eyeball, presented no symptoms whatever because of the conditions that existed.

Examination showed the right eyeball to be proptosed downward, outward, and forward (Figs. 1 and 2). The proptosis was marked, but the eyelids could be entirely closed. The rotations of the eyeball were concomitant with those of

the other eye, but because of the proptosis the right eye could be rotated further downward than the left. As previously stated, no symptom other than a dull eye-ache which appeared occasionally was observed by the patient. She complained only of the proptosis from an esthetic standpoint.

The vision of the right eye equaled 6 6, and the patient could read easily J.1 at six inches. Vision of the left eye equaled 6/5, and she also could read J.1 easily at six inches

Fig. 1.—Proptosis of right eye.

Fig. 2.—Lateral view.

with this eye. There were three degrees of esophoria. No diplopia could be elicited either with or without the red glass. Abduction, 4°; adduction, 15°.

The pupil of the right eye was 4 mm. in diameter and somewhat more sluggish in its movements than that of the left eye. The left pupil was 2½ mm. in diameter. Both reacted promptly to light, convergence, and accommodation.

The optic nerve of the right eye was slightly paler than normal, but there were no indications of any preëxisting neuritis.

Examination of the orbit revealed a large bony growth which could be distinctly felt with the fingers in the upper inner portion. It extended transversely from the region of the ethmoid about half way across the roof of the orbit toward the temporal side, and in depth it passed backward toward the apex of the orbit as far as the finger could feel.

The examination of the nose revealed a small amount of pus in the right naris. None was discovered in the left. The patient did not complain of any sinus symptoms, nor were there any indications of occlusion of the sinuses from intranasal pressure. Transillumination showed a very small frontal sinus on the left side, the area of the right frontal sinus being entirely dark. This indicated, of course, an absence of the sinus or some affection which prevented transillumination. The upper portion of the right antrum just below the orbit also seemed slightly darker than that of the left side, but exploratory puncture brought no pus.

The field of vision for the right eye was practically normal, and there was no scotoma for form or color. The urine examination was negative. No Wassermann was made.

Skiagraphs made by Dr. W. W. Potter show not only the orbital portion of the skull to be involved, but that a bony growth as large as a medium-sized hen's-egg extends into the cerebral cavity in the anterior cerebral fossa, giving a shadow 58 mm. long and 25 mm. broad, measuring from the shadow produced by the base of the skull. This, of course, does not include the concavity of the cerebral fossa, which means necessarily that the growth is much larger than above indicated. The growth extends from the frontal sinus anteriorly to the sella turcica posteriorly, overlapping the latter several millimeters. The skiagraphs (Fig. 3) would seem to indicate that the frontal, ethmoidal, and sphenoidal sinuses on the right side were all involved.

It is believed, therefore, that the condition is one of osteoma, in all probability having its origin in the frontal sinus, both the orbit and the anterior cerebral fossa being involved to the extent above described. While it is a defin-

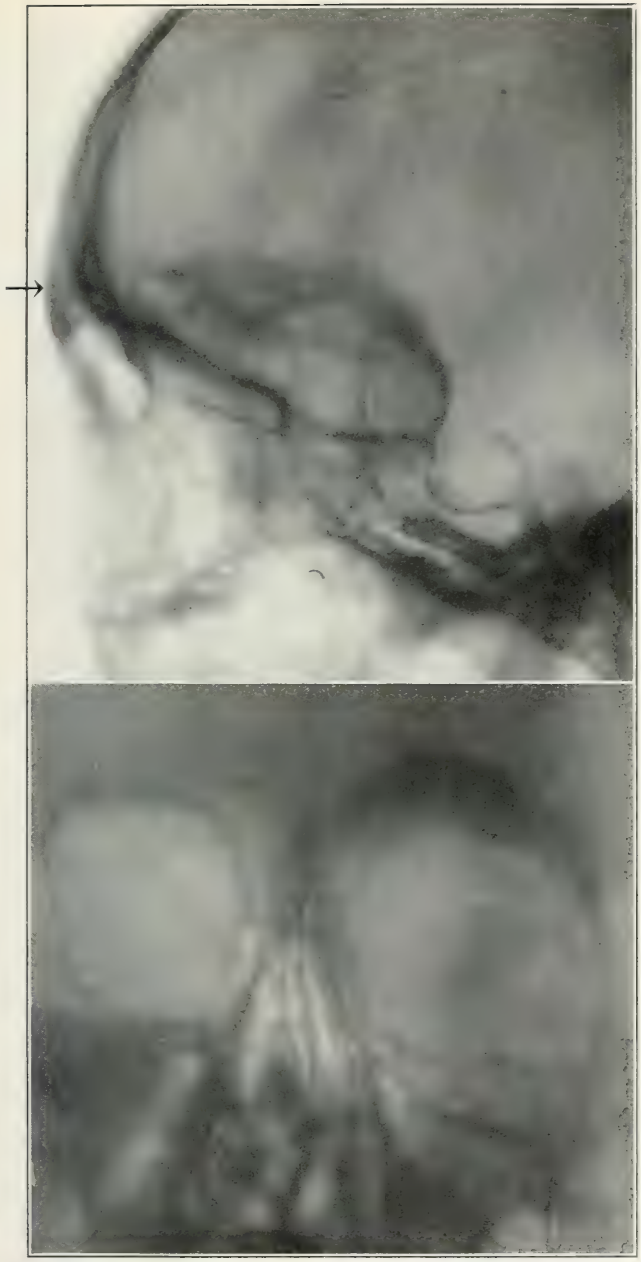


Fig. 3.—Skiagraphs giving lateral and front views of skull.

itely established fact that injuries to the anterior portion of the brain or pressure in this same region is less likely to produce severe disturbance than injury or pressure to the same extent in some other portion of the cerebral cavity, it appears to the writer most unusual that a bony growth of the size of that described, and pressing so deeply into the base of the brain in the anterior portion and extending backward to a point slightly beyond the anterior wall of the sella turcica, could exist without producing a single symptom of cerebral pressure.

Inasmuch as the patient complained only of the proptosis, and there were and had been no other symptoms, the extent of the growth into the cerebral cavity seemed to the writer to preclude the advisability of any operative procedure at the present time. Should symptoms of cerebral compression arise at any future date, some operative procedure as a palliative measure would, of course, have to be undertaken.

DISCUSSION.

DR. R. A. REEVE, Toronto, Canada: It was my good fortune to be present at Moorefield, during the session of the International Medical Congress in London in 1881, and to see Sir John Tweedy attempt the removal of an osteoma which apparently, from the description, corresponds very closely with that described by Dr. Veasey. Sir John Tweedy contented himself with the removal of the orbital portion by means of the chisel and mallet, and the patient apparently recovered very well, but about four weeks later developed serious symptoms of deep metastasis and died. At the time of the operation Dr. Herman Knapp was present, and it was my privilege to inspect the hospital with him later, and he made the remark: "Dr. Reeve, that is the event of this Congress."

DR. EDWARD JACKSON, Denver, Colo.: In a case I reported some years ago, not so extensive as that reported by Dr. Veasey, but which seemed as if it might extend into the anterior cerebral fossa, a portion of the bone was first re-

moved, but finally the whole was enucleated from the frontal sinus. That patient I saw after five years with no signs of extension or recurrence, and I believe she is still living and in good health. The displacement of the eyeball was quite as great in that case as in this; and the removal was followed by retreat of the eyeball to normal position. The disturbance of movement, previously existing, and the diplopia resulting from the retreat of the eyeball immediately after the operation, had passed away in a very few days.

TWO CASES OF ORBITAL GROWTH—(A) MIXED TUMOR OF THE LACRIMAL GLAND. (B) SMALL ROUND-CELL SARCOMA IN AN INFANT.

T. B. HOLLOWAY, M.D.,
Philadelphia.

Since new-growths of the lacrimal gland are but infrequently encountered, the data submitted below seem of sufficient importance to place on record. The second case referred to well illustrates the early fatal termination of those cases of rapidly growing small round-cell sarcoma in infants.

CASE I.—Mr. H. W., aged thirty-five years, was referred to me by Dr. George Dorrance, of Philadelphia, on June 23, 1915. In the upper and outer portion of the left orbit some fullness was first noted seven years ago. Inside of a year the eye had become prominent and the vision distinctly impaired. At this time he was told by an oculist, with whom he remained under treatment for some six weeks, that the vision of the affected eye was one-sixth of normal. Since that time the proptosis has gradually become more marked, but there has never been any pain or acute inflammatory affection of the eye.

At the time of examination the right pupil was round, the left slightly oval, but both reacted promptly to direct light.

Fig. 1.—Case I. Before operation.

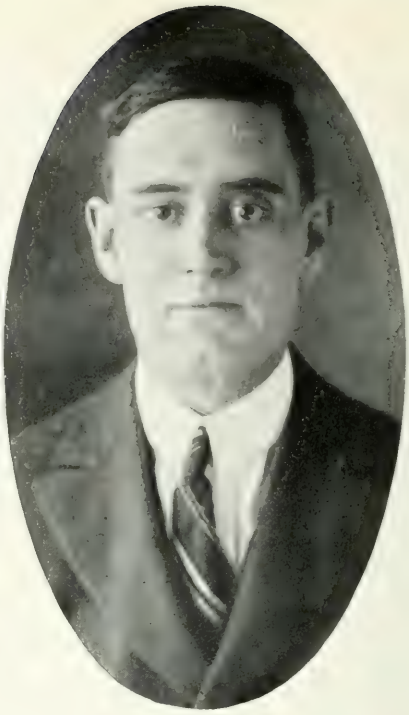


Fig. 2.—Case I. Four months after operation.

Fig. 3. –Case II. Involvement right orbit after evisceration of left.

Fig. 4.—Case II. Eight days before death.

The right eye failed to show any abnormalities either externally or internally. The left eye was markedly proptosed, the globe being displaced downward 6 mm., inward 4 mm., and with Hertel's exophthalmometer the prominence of the left eye measured 27 mm. as compared with 17 mm. for the right eye (Fig. 1). Pressure on the globe backward met with marked resistance. The lower margin of the orbit was free. Beginning just internal to the midline of the upper orbital margin and extending to the outer canthus, between the globe and the margin of the orbit, was a hard, lobulated growth measuring approximately 3.75 cm. in length and 2.5 cm. in width. The width included what was apparently the orbital margin that had been rounded off by being everted and turned backward anteriorly. Intraocular examination showed the media to be clear, the disc slightly oval, with rather hazy yellow margins; the nasal half was distinctly hyperemic, which contrasted rather sharply with the temporal portion, which was but little less than average capillarity. In the macular region there was a superficial retinochoroidal disturbance associated with delicate punctate pigmentation. The veins were full but not engorged; the arteries were not contracted, and there were no free hemorrhages or exudates. The form field of the left eye was full, with decided contraction of the blue and red field; green was not recognized. At fixation there was a thin negative scotoma. V. R. E. = 6/5; L. E., = 6/100.

The patient was not seen again until October 28, 1915, when no appreciable changes had occurred. In the interval between these visits an *x*-ray examination had been made by Dr. Thissell at the office of Dr. Dorrance, and he reported a shadow in the left orbital region, but believed that this was not dense enough for bone. Alteration in the orbital margin was also noted. Diagnosis: Probable mixed tumor of the lacrimal gland.

Having obtained consent to enucleate, or eviscerate if necessary, the patient was subjected to operation on December 1, 1915. Beginning just external to the supraorbital notch, a curved incision 4.5 cm. long was made through the site of the eyebrow. By careful dissection the external portion of the growth was exposed and the whole mass was completely freed by blunt dissection. No incisions adjacent

to the tumor were made except through the overlying structures. After removal of the mass, which was rather tedious, owing to the limited incision and the length of the growth, the orbit was exposed practically back to the optic foramen. The orbital fascia was then firmly brought together by interrupted catgut sutures, deep silk sutures being used for the cutaneous wound. The convalescence was uncomplicated and he was discharged on the tenth day.

January 12, 1916: The right palpebral fissure measured 10 mm.; the left, 11 mm. With the exophthalmometer O. D. measured 17, while O. S. was 18.5 mm. The right eye could be rotated outward to within 1 mm. of the external canthus; the left eye to within 2 mm. of the canthus. There was no impairment in rotations upward, inward, or downward, and the lids could be forcibly closed. V. = 6/50.

The patient was last seen April 13, 1916, when the prominence and rotation of the globe remained about the same (Fig. 2). The intraocular conditions remained as above described, except that the tint of the disc tended to be more uniform, owing to lessened capillarity of the nasal portion. V. = 6/50.

Pathologic Report by Dr. Allen J. Smith.—Specimen consists of a rounded, hard, nodulated tumor 40 x 35 x 20 mm. It is yellowish gray externally, well circumscribed, and apparently has a very thin capsule. Cuts with resistance. Sectioned surface shows a gray-white periphery and yellowish, granular, but firm center.

Section made from a part of the growth including the periphery shows the tumor to have been inclosed in a well-developed and at places hyalinized fibrous capsule. The growth proper is relatively avascular and apparently primarily an epithelial neoplasm which has grown for the most part as an adenocarcinoma. At places there is reversion of the glandular type of epithelium to squamous type, and here and there epithelial pearls, such as are found in squamous epithelioma, are to be noted. A mucinous secretion from the glandular elements has given rise secondarily to a number of special features. For example, at places it lies as ordinary hematoxylin tinted mucin in small cysts. In other places it has been chemically so altered as to take on the eosin hue (a pseudomucinous change), and resembles

Fig. 5.—Case I.

Fig. 6.—Case I.

hyaline material (giving the appearance mentioned in Ziegler's pathology under the name cylindroma carcinomatodes). Moreover this material, at places as ordinary mucin and elsewhere more like hyaline substance (pseudomucin?), has escaped into the stroma, infiltrating the latter or forming larger collections in the stroma. The latter type, when including cellular elements, as it often does, histologically closely resembled the irregular cartilage of a chondroma, and is probably the basis for reports of true chondromata of these glands; but it is not to be detected grossly as cartilage, and in the light of what is above said is not cartilage. Some simulation at a few places of bone are also to be seen, but this is only histologically apparent and not real bone.

In the rather scant fibrous stroma the epithelial cells of this cancer have infiltrated; and, while undoubted epithelial cells appear at places, are often so altered as to appear like young connective-tissue corpuscles and thus the appearance of large round-cell sarcoma (or endothelioma) develops, which has also been noted by the writer as an element of "mixture" in these so-called "mixed tumors of the lacrimal glands." The gradation of the clearly epithelial cells into this type of element and the relative avascularity are regarded by the writer as sufficient to refer these apparent foci of sarcoma to the cancer itself. Finally, from the diffusion of the mucin in the stroma at places an appearance of a myxoma is added; but again, in the writer's opinion, not as a true myxomatous admixture, but as a mechanical artefact from the escape of the mucin into the tissue (Figs. 5 and 6).

The growth is an example of so-called "mixed tumors" of the lacrimal glands, and might, by mistake, have been called a fibro-myxo-chondro-osteo-cylindro-sarcoma carcinomatodes; but the writer, as above suggested, prefers to look upon it as an adenocarcinoma with reversionary, infiltrative, and degenerative changes.

CASE II.—D. R., aged fourteen months, was referred to me on October 9, 1914, by Dr. I. B. Roberts, of Llanerch. The infant was well nourished and, aside from lack of color, appeared healthy. The mother stated that the birth was

normal, the infant breast fed, and had never had any diseases, "not even a cold." On September 10th the child fell down several steps, but was apparently unhurt and was not marked. Later the mother noted slight redness and swelling of the left lower lid, which gradually increased during the next four days with the involvement of the upper lid. There was no indication of pain. On September 18th the child was seen by a physician, who suggested the possibility of an insect-bite. The condition gradually became worse, with injection of the bulbar conjunctiva, and when next seen by her physician on October 5th he detected slight prominence of the globe. She was first seen by Dr. Roberts on October 7th.

Externally the right eye was normal. The upper and lower lids of the left eye, while reddened, presented a distinct bluish discoloration, more marked in the lower lid. The veins of the lids were moderately dilated. The globe was markedly proptosed and was displaced upward about 8 mm. The chemosis of the lower bulbar conjunctiva was sufficient to project between the lids, and was covered in places by thin crusts; the upper bulbar conjunctiva was but slightly injected. The cornea was clear. The upper margin of the orbit was free from any growth or thickening, but beginning at the external canthus and sweeping along the lower margin could be felt a firm, resisting cylindric mass, not adherent to the skin. There was firm resistance to backward pressure of the globe. The following day an *x*-ray examination was made by Dr. Pancoast, but nothing of positive value could be determined. While under anesthesia a fundus examination was made which revealed nothing more than hazy disc margins, with full and tortuous retinal veins in the left eye.

Dr. G. E. de Schweinitz saw the patient in consultation and concurred in the diagnosis of orbital sarcoma. Dr. J. P. Crozer Griffith made a careful examination as to the child's general condition, but failed to find any abnormalities. Blood examination: Red cells, 3,450,000; white cells, 8,800; hemoglobin, 58 per cent.

On October 16th the left orbit was eviscerated and the lids sutured, with the insertion of a small drain. Five days later a tube containing 25 mgm. of radium was suspended in

the orbit for eight hours. There was no external reaction, but there was an increase in the discharge, due to breaking down of the clot. On November 10th slight edema of the right upper lid was noted. An examination made by Dr. Charles Fife revealed slight dulness at the base of the right lung, and the liver was distinctly palpable 3 cm. below the costal margin. From this time on the right eye developed all the changes that have already been described as affecting the left eye (Fig. 3). Distinct fullness and swelling developed over the right antrum; there was a recurrence in the left orbit, and fullness and swelling above and below the zygoma in the temporal region. The patient was removed from the hospital on December 2d (Fig. 4) and died one week later. No autopsy was permitted.

Pathologic Report by Dr. Allen J. Smith.—Section of the growth shows it to be made up, with fair uniformity, of small, round, connective-tissue cells, with proportionately large nuclei and scant cytoplasm, the nucleus of each rich in chromatin and deeply staining. There is but little intercellular material. The blood-vessels are not numerous, and when noted are of capillary type (endothelial-lined channels). There is no pigment save in a few isolated places, and here of stagnatory type rather than melanin. Small areas of hemorrhage are encountered, probably the result of artefact. The growth in different sections is seen in its mass infiltrating the orbital fat and the ocular muscles. Diagnosis: Small round-cell sarcoma.

Sections of the globe failed to show any invasion of the ocular tissues. No structural changes were noted in the cornea, iris, ciliary body, and none in the lens, save from artefact. Aside from a fibrosis of the vessels, the retina appeared normal. A small fragment of the optic nerve was met in one of the sections. This showed a slight perivascular cellular infiltration, fibrosis of the larger vessels of the nerve, and apparently a dilatation of the subdural lymph-space.

It will be recalled that Warthin,[1] in 1901, after an analysis of 132 cases found in the literature, called attention to the multiplicity of terms used to describe certain growths of the

gland, and that in all probability these represented but different phases of the same type growth, that is, the so-called mixed tumor. Greeves states that the earlier suggestion of Dianoux as to the similarity of growths of the lacrimal and salivary glands had not been generally recognized. Warthin's painstaking paper was shortly followed by the excellent contribution of Verhoeff,[2] who reviewed five cases of "mixed tumor," four of which came under observation at the Massachusetts Charitable Eye and Ear Infirmary during a period of ten years. While this is the most common tumor of the lacrimal gland, its frequency as found by Verhoeff was one among 50,000 ophthalmic patients. Six years ago Jack and Verhoeff[3] reported the data of another case before this society, and, with this patient, two orbital operations were followed by local recurrence and death. In 1914 Greeves,[4] after a review of the literature from the time of Warthin's report, analyzed 42 cases of tumor of the lacrimal gland. He divided these into two groups: Class A, mixed tumors; and Class B, tumors the main histologic feature of which is an overgrowth of small round-cells in the gland stroma. Class A, which comprised 24 cases, he divided into groups conforming to three different types, while Class B, which comprised 18 cases, has been subdivided according to whether but one lacrimal gland was affected or whether one lacrimal was associated with an involvement of one salivary gland, or finally, whether there was a simultaneous involvement of the lacrimal and salivary glands.

Concerning the histogenesis of mixed tumors, various views have been expressed. In Greeves' splendid study of these growths a concise résumé of the opinions of various investigators will be found. Still more recently Birch-Hirschfeld[5] has placed on record the history of one of these cases, and has reviewed the literature of 75 others. He regards the parenchyma cells as of epithelial rather than of endothelial origin. To emphasize the congenital origin of these

growths, as well as their epithelial nature, Verhoeff has suggested the term of congenital epithelioma.

Concerning Case 1, it is of clinical interest to point out the decided deformity of the upper and outer margin of the orbit, dependent upon the long-continued pressure of the growth. Doubtless there was some prominence of the globe for a long period of time before it was appreciated by the patient. This deformity is much more easily determined since the removal of the growth, and, as has been stated and as would be expected, the margin is curled anteriorly. This deformity, as well as the line of incision, is now well concealed by the development of the eyebrow. This altered orbital margin is doubtless responsible in part for the greater measurement on the left side with the exophthalmometer, but aside from this the globe is very slightly more prominent than its fellow, although there is no vertical or horizontal displacement and no resistance to backward pressure. I can explain this slight persistent prominence only by assuming a loss of tone in the muscles and fascia as the result of the prolonged extreme tension on these structures.

As to the second case, the picture presented was one that can readily be recalled by all who have had experience with these highly malignant growths in infancy. Doubtless there was some involvement of the antrum at the time of evisceration. The local recurrence, involvement of adjacent structures by extension, and the probable existence of metastases that an autopsy would have doubtless revealed, present a course that is as typical as it is hopeless.

REFERENCES.

1. Warthin: Arch. of Ophthal., 1901, xxx, p. 601.
2. Verhoeff: Journal of Medical Research, 1904–5, viii, p. 319.
3. Jack and Verhoeff: Trans. Amer. Ophthal. Soc., 1910, xii, pt. 2, p. 414.
4. Greeves: Royal London Ophthalmic Hospital Reports, 1913–14, xix, p. 237.
5. Birch-Hirschfeld: Arch. f. Ophthal., xc, p. 110; Ophthalmic Literature, 1916, vi, p. 27.

RECURRENT SARCOMATA AT THE LIMBUS TREATED BY "ELECTRIC DESICCATION."

BURTON CHANCE, M.D.,
Philadelphia.

In whatever aspect we view them, be it their origin, their pathologic significance, or their treatment, tumors of the anterior segment of the globe are always of interest. To-day I wish to report two cases of epibulbar sarcoma at the limbus in which there were recurrences of the tumors after surgical removal, and to advocate a method of treatment which shall have, I hope, not only the merit of removing the active tumorous processes, but also the prevention of their recurrence.

A railway engine driver, aged fifty-eight, consulted me in May, 1915, because he had been urged to have his left eye excised by reason of the rapid growth of a tumor at the sclerocornea. The tumor had appeared as a tiny dark speck in July or August of the previous year; he had regarded the effects of a bruise from a flying piece of coal as the cause of it. It had been treated by a variety of methods, all of which only stimulated rather than checked its growth. The man was in vigorous health; he had never had piles nor growths elsewhere.

The tumor mass, of the size of a small bean, projected through the palpebral fissure. The base of the tumor was much smaller than the circumference of its body; it arose by a narrow stalk at the sclerocorneal junction in the lower temporal quadrant, and extended well on to the cornea, to which it was more superficially attached than it was to the conjunctivosclera, yet it was so firmly adherent to the underlying tissues that it could not easily be moved. The mass was nodular and more or less fungoid, except at the

portion pressed upon by the upper lid, the surface of which was quite smooth. It was not friable, neither did it bleed readily; indeed, the tumor itself seemed to be bloodless, for it was pearly white in color, although many large and tortuous vessels from the conjunctiva converged at its base. The cornea otherwise was not affected, nor were there other suspicious spots about the limbus, and the conjunctiva was not at all discolored.

The man's sight had been not at all affected, but was at the full standard of acuity, and the fundi were healthy except for such changes as constitute "the fire-worker's choroids."

On the next day I excised the tumor by a dissection carried well out into the bulbar conjunctiva and over the cornea, although the base of the tumor had not penetrated the corneal substance. The conjunctival tissues were further loosened, and the raw surface covered by uniting their edges by four sutures. The tumor did not bleed, although the hemorrhage from the large vessels was quite free. All the sutures came away at the end of the fifth day; the healing was so rapid that in three weeks' time the region appeared normal, except for the quite wide dilatation of the conjunctival vessels.

In July, however, the eye became red again, and there was a granular and vascular mass occupying exactly the same site as that of the excised mass, but extending somewhat more widely than it had. This growth, too, was nodular and fungoid, and there was a greater degree of vascularity of the conjunctiva than was present previously. This mass was excised August 16th. It appeared to have gone somewhat more deeply into the corneal tissue than was the case with the first mass. The surface healed readily; but after two weeks a small nodule again appeared.

On September 24th there was an exuberant, double-headed mass; this, too, was duly excised, and four weeks later the region seemed to be entirely free. But in eight weeks, by November 27th, the man stated that for two weeks his friends had remarked the reappearance of a tumor mass.

A study of the three separately excised masses showed them to be sarcomata.

I did not disturb this last mass until December 31st, by which time it had become quite as large as that which was

first excised. Still another nodule was present by January 22, 1916, and of an even greater exuberance. In addition, and for the first time, the man complained that the eye was painful. By February 8th this last mass had attained a size greater than any yet seen; besides, it protruded prominently through the palpebral fissure (Fig. 1).

I determined not to attempt any further excisions, but, from my observation of the results attained by the desiccation of neoplasmata of the lids and cheeks at the hands of Dr. William L. Clark, I believed such a method might be used on the globe as well. Accordingly, I took the patient to Dr. Clark, who, under my direction, seared the mass, slightly at first, scraping away the slough which followed, after which the surface was seared twice again, but the last slough was not disturbed. The Medusa-like vessels were then "punctured" with a tiny flame, at points well back into the fornix, which had the good effect of obstructing their lumina.

When dressed the next day, the reaction was found to be slight. There had been but little hemorrhage during the operation, but now the temporal half of the cornea appeared brilliantly blood-stained and the site of the tumor coagulated. The patient returned to his home in central New York, but visited me after six days. In the interval the area had healed entirely, and, except for the blood-staining, the corneal surface was healthy. On March 24th the part where the tumors had been appeared to be only slightly coagulated, and in place of the former blood-staining there was a murkiness which extended into the cornea. The large vessels had become contracted, and the eye was as quiet and as natural looking as its fellow; it was not at all tender nor painful (Fig. 2).

Dr. P. N. K. Schwenk most generously wishes me to report from his service at the Wills Hospital, Philadelphia, the case of a melanosarcoma at the limbus that recurred after excision, which new-growth I recommended should be subjected to desiccation.

A woman, aged thirty-seven years, came to the hospital in October, 1914, because of a comparatively large mass on

Fig. 1.

Fig. 2.

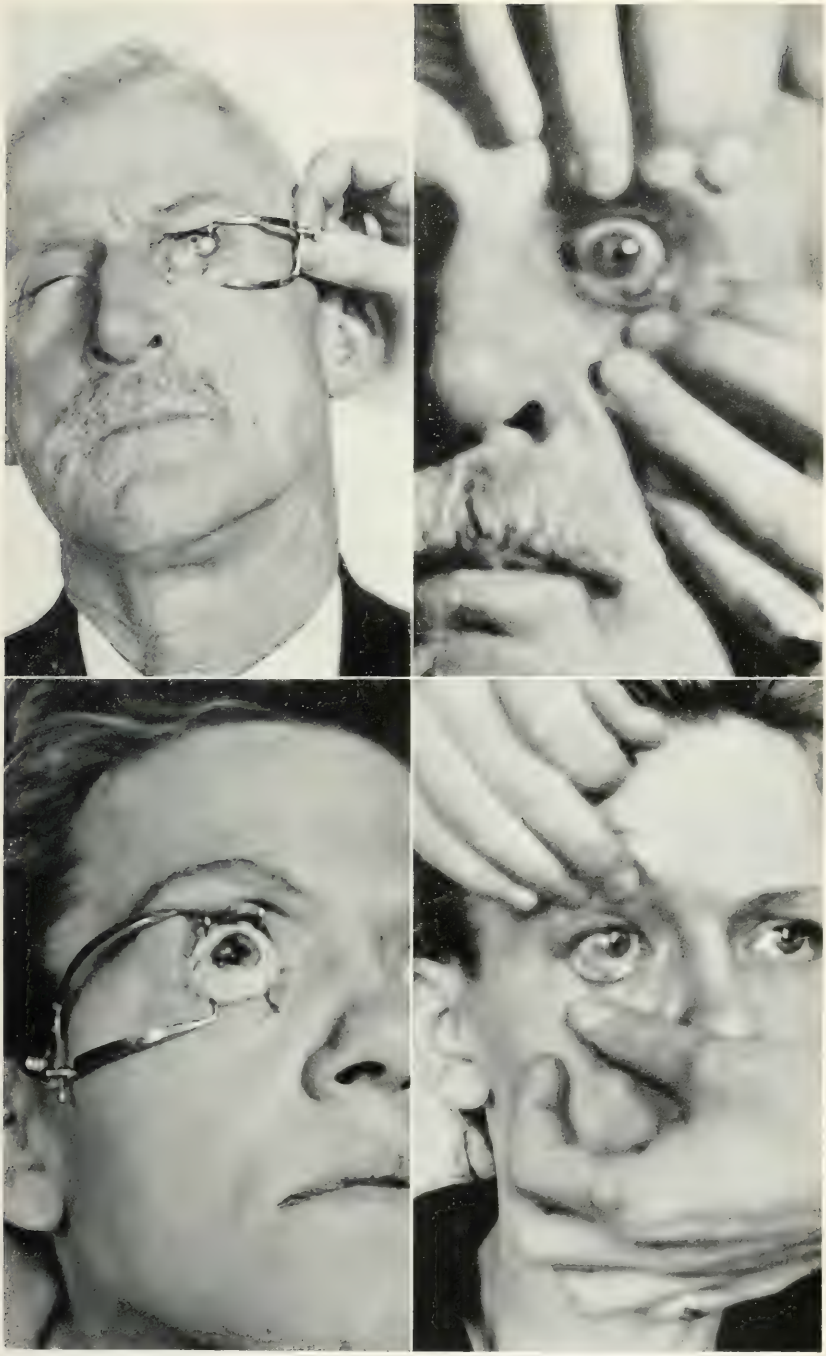


Fig. 3.

Fig. 4.

her right eye. She stated that from her earliest infancy there had been a dark spot on the white of the eye, a short distance from the clear part, which spot had grown gradually until shortly after the first of her six children was born, when it perceptibly increased in size; and at each successive pregnancy it had become larger than it had been in the intervals. When she was examined by us there was a kidney-shaped mass about 6.5 mm. by 2.5 mm. in the chief diameters lying well over the temporal side of the cornea, without protruding through the palpebral fissure. It arose beyond the limbus and seemed to be adherent to the cornea; the remaining portion of the cornea was clear, and the iris healthy. By reason of its grayish-brown color it resembled a huge uveal prolapsus. Quite large blood-vessels converged toward it from the scleral conjunctiva. There were no disturbances noticeable within the fundus. V. R. E. = 6/12; L. E. = 6/7.5.

On closer inspection the mass was found to be not at all adherent to the cornea, but being attached by a short pedicle it could be lifted up; the pressure of the lid had flattened it against the cornea. It was excised on November 14, 1914, and the recovery was prompt. The patient continued under observation for several weeks, but as there seemed to be no further need for attendance, she disappeared. An examination of the mass showed it to be a pigmented sarcoma.

On February 17, 1916, she returned because of a reappearance of the growth. She was not certain when the first signs of the recurrence had appeared, but in the previous three months it had grown rapidly, although she was not again pregnant. The mass, which was quite vascular, occupied the same site as the original growth; it seemed to be exactly like the first tumor, of a grayish-brown color, and covered with conjunctiva (Fig. 3). On February 8th she was referred to Dr. Clark, who, according to directions, but without my presence, "desiccated" the mass. The result has been ideal, although for a few days the cornea in the region of the tumor showed coagulation, but not any blood-staining. When seen within the past four weeks the surface formerly occupied by the tumor was smooth and perfectly healthy (Fig. 4).

By "desiccation" is meant the dehydration of tissues by means of heat applied in the form of an electric "flame" produced by a high-frequency current, whereby the vitality of tissue-cells is destroyed. The heat is produced by a monopolar electric current of high tension, generated best by a static machine revolving at great speed, and then transformed by suitable appliances. The heat-flame, which must not be so intense as to char the tissues, flows from the point of a fine steel needle. The current from such an instrument can be regulated precisely, and the visibility of the flame affords one the opportunity to apply it exactly when and where the operator desires.

This is not the so-called "fulguration," which consists in the indirect destruction of tissue by the application of a current generated by an induction coil or transformer, through the action of which the nutrition of a part is so altered as to prevent the proliferation of cells.

The technic employed by Dr. Clark consists very much as follows: Local anesthetization by cocain with adrenalin, either by instillation or by infiltration. A fine needle is fixed in a suitable hand-piece properly insulated. The current is made and broken by an assistant on signal from the operator, whose attention should not be distracted from the field of operation. The thermic intensity of the "flame" is tested by approximating it to a bit of metal, such as the point of the forceps. No arbitrary rules can be given for the application of the "flame," because different cases require different strengths. One's technic develops with one's practice. The needle is not usually thrust into the tissues, but is brushed over the surface of the mass to be removed. The depth of the desiccation depends upon the length of the time of the contact and the driving power of the current, which, of course, can be adequately regulated. It may not be required extensively and visibly to destroy all the area that the heat penetrates, but only sufficiently to cause a retro-

gression and disappearance of the tissue-cells. In the case of growths in the anterior segment of the globe the cornea should be protected by means of a metal spatula to prevent the coagulation, if not the destruction, of that membrane.*

After the tumor mass has been converted into a necrotic heap it is to be curetted away, or it may be excised. It might be well, in the case of a conjunctival growth, to allow the mass to macerate and slough; which event proved a rapid and bloodless process in each of the cases here reported. Infection cannot readily occur on such a seared surface because the blood-vessels and lymph-channels have become sealed. Dr. Clark has not seen infection follow in any case under his observation. The healed surface presents neither scar, distortion, nor other cicatrizing process.

It is hardly proper to presume to dictate that this method of treatment should be pursued in all cases of neoplastic processes of the ocular tissues just because of the immediate results of the application of the method to the two cases here presented, yet from the results which I have observed in a variety of cases so treated by Dr. Clark I would advocate the use of it, rather than the customary surgical procedures, because it has seemed to have checked recurrences and allayed, rather than excited, cell-formation, the contrary results having been so frequently the case after the excision of new-growths and notably manifested in my first patient. The blood-vessels and lymph-channels become sealed by the heat. And, as no cicatrix nor distortion follows, because only unhealthy tissues are to be subjected to the heat, it need not matter, even though desiccation should have to be repeated by reason of the recurrence of the neoplasm.

Desiccation possesses distinct advantages over radium and other emanant rays, in so far that the flame can be seen and

* Clark, W. L.: Desiccation Surgery: Medical Council, July, 1915, in which are to be found numerous bibliographic references.

determined at the time of the application; its action is constant; and it can be confined to a definite area so that the surrounding tissues need not be harmed, nor should the sight be damaged by its use.

DISCUSSION.

DR. GEO. S. DERBY, Boston, Mass.: I understood Dr. Chance to say that the heat was determined by approximating the needle to a piece of metal, and I would like to know exactly the method by which it is determined.

DR. CHANCE: I meant by that statement that the electric flame, or spark, is caused by the contact of the needle with an object, and, of course, one can readily gauge the size and power and make it long or short, depending upon the distance between the needle and the object, and dull or brilliant according to the power of the generator. In these two cases I should say the flame, or spark, was moderately brilliant, but was not more than one-eighth of an inch in length; for deeper areas, however, the operator would make a longer and more intense flame.

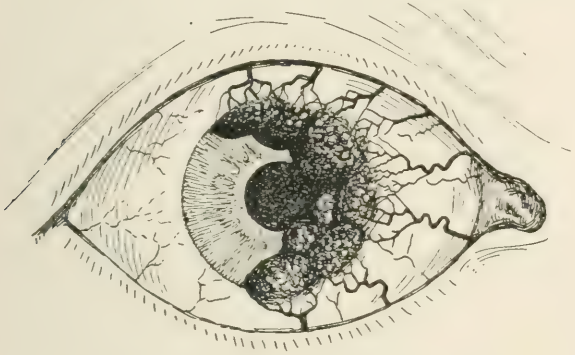
AN EPIBULBAR MELANOSARCOMA OF THE LIMBUS WHICH COMPLETELY DISAPPEARED UNDER *X*-RAY TREATMENTS.

ALBERT C. SNELL, M.D.,
Rochester, N. Y.

Gerardo DeCarolis, an Italian, aged thirty years, first consulted me September 15, 1913, because of a growth on his right eye, which, he stated, he had first noticed five years before. He said that he had been treated by several different oculists, and that the growth had been removed on five different occasions, the last July 8, 1913, by Dr. Frank Barber, of Rochester, N. Y., who removed the major portion of the mass.

In a communication Dr. Barber describes the tumor as seen by him at this time as follows: "Above and to the

nasal side, extending from the conjunctiva over the limbus and a little way into the cornea, was a dark-brown, sausage-shaped tumor about 3 mm. wide and 5 mm. long. Extending from the tumor backward into the superior fornix were two or three large vessels. The color was rather striking, as it was a peculiar seal-brown and gave one the impression of iron rust. A piece was excised for microscopic examination and unfortunately lost. Two weeks later the tumor had reached a larger size than when it was first removed. It was again removed, and the base thoroughly curetted. A section was submitted to Dr. Daly, and he reported that it was a melanosarcoma of round-cell type. Subsequently



Epibulbar melanosarcoma of the limbus.

the patient admitted that the growth had been removed by an oculist during the preceding March. From the rapid recurrence of the tumor a bad prognosis was given, and the patient advised to have the eye enucleated if it recurred again."

At the time of my examination this growth occupied two-thirds of the inner corneoconjunctival limbus, lying almost entirely on the cornea, and reaching the inner edge of the pupil, slightly covering it. The mass was firmly adherent below, although the outer edge slightly overlapped the subjacent corneal tissue. The entire mass was dark seal-brown in color, the upper half being slightly darker than the lower. It was divided into eight distinct lobules, differ-

ing but little in size, each lobule having a nodular but unbroken surface. The entire mass was roughly crescentic in shape, the periphery of the arc measuring 26 mm. The crests of the lobules reached different elevations, being about 2, 3, and 4 mm. high. Entering the mass from the nasal and the superior bulbar conjunctiva there were eight dilated, branching blood-vessels. The nasal third of the cornea was perfectly clear; the pupil reacted promptly to light stimulus, and the ophthalmoscope revealed no alteration from normal in any of the fundus structures. V. = 20 20.

It occurred to me, in view of the fact that this growth was apparently superficial, that *x*-ray treatments might be of value. Therefore, after consultation with roentgenologist Dr. M. B. Palmer, such treatments were instituted. Altogether 12 treatments were given, the first October 31, 1913, the last January 31, 1914.

Dr. Palmer describes the technic employed thus: "The tube used was a protected lead-glass diaphragm, tube being placed as close to the eye as the glass cone would permit, which was about five inches from the target. A medium soft tube was used, two to four minutes being the time of exposure. (Dosage not measured at this time.) A saturated solution of boric acid was kept constantly flowing over the cornea during each treatment. Improvement was noted after the fourth treatment. Treatments were discontinued when two-thirds of the area was healed."

Six weeks after the last treatment the entire tumor had completely disappeared. The cornea was smooth, perfectly clear, and had resumed its normal contour. At the limbus, and extending into the conjunctiva beyond, there were several irregular islands of pale brownish pigment deposits, some 1 x 2 mm., others mere dots. These pigmented areas were not elevated, and there was no macroscopic evidence of any thickening of the conjunctiva or of the presence of any blood-vessels. The man was seen again three months later, at which time there was no evidence of any recurrence. In August, 1915, a friend and fellow-workman reported that DeCarolis had had no more "eye trouble." I have been unable to locate him since. He was told of the danger and the possibility of a recurrence, and he promised to see us if this happened, but he has not been heard from.

Since the classic paper by Verhoeff and Loring,[1] many cases of epibulbar sarcoma of the limbus have been reported in the literature. Among many others the articles by Parsons,[2] Clegg and Hall,[3] and recently one by Crigler,[4] review the literature to recent date.

The case which I have herewith reported was of the usual type of melanotic, epibulbar sarcoma of the limbus. Dr. Edward A. Shumway kindly examined the slide and writes: "The cells are of the mixed round and polygonal shape, which are usually found in these tumors, and in the little nevi from which they frequently develop. They are arranged in alveoli, and separated by bands of connective tissue. The amount of pigment is not great, so that the cellular structure is not concealed. There is also a moderate amount of round-cell infiltration in parts of the tumor, indicating an inflammatory reaction of the tissues. As you know, these growths are considered by some to be of epithelial origin, but I personally believe they are mesoblastic, either arising from the connective-tissue elements or from the endothelial cells of the tissue."

The principal point of interest in this case is the result obtained from Roentgen therapy. In the literature I found but a single case, that reported by Braunschweig,[5] in which *x*-ray treatment had been used. In this case treatment was given after complete excision, and there was no recurrence for three years. In our case the treatment was undertaken at the time the tumor had reached its maximum growth and without an excision preceding the treatment; and, so far as I am able to discover, it is the first case treated solely by *x*-rays.

In view of the facts presented in this case that, although the tumor had recurred soon after removal, having been removed five times during a year, and each time after removal recurring more rapidly and becoming larger, the tumor rapidly disintegrated under *x*-ray exposures, dis-

appearing entirely six weeks after the last treatment, without recurrence at least in one year and possibly in two, it seems to me that we should consider the employment of Roentgen therapy in the treatment of some of these cases, especially those in which the ciliary body has not become involved.

REFERENCES.

1. Verhoeff and Loring: Arch. of Ophthal., xxii, 1903.
2. Parsons: Pathology of the Eye, i, p. 138.
3. Clegg and Hall: Lancet, September 10, 1904.
4. Crigler: Arch. of Ophthal., xliv, p. 41.
5. Braunschweig: Klin. Monatsbl. f. Augenh., February, 1908.

REPORT OF A CASE OF MELANOSARCOMA OF THE ORBIT TREATED WITH RADIUM.

EDWARD B. HECKEL, M.D.,
Pittsburgh, Pa.
(From the Service of the Allegheny General Hospital.)

The subject of the following observation, a man aged forty-eight years, was seen first on April 16, 1912. There was no history of any eye injury; general good health had always prevailed. The right eye had never annoyed him; the left eye, however, was and had been blind for twelve years; the condition then present had commenced thirteen years before: "it simply got weak."

Inspection showed the right eye negative in every respect, with a corrected vision of 20 20 with +0.75 D. c. ax. 180°. Left eye: the cornea flat, yellowish, and opaque, with some blood-vessels in its substance. It appeared like an eyeball which had undergone a sloughing of the cornea following a purulent conjunctivitis, although the patient denied any inflammatory condition of the eyeball at any time. The eyeball was somewhat shrunken and soft. An immediate enucleation was advised, but was refused by the patient, as he had heard "the removal of one eye would injure the other."

Fig. 1.

Fig. 2.

Fig. 3.

Melanosarcoma of the orbit treated with radium.

The patient returned, however, on September 9, 1913, for an enucleation of the left eyeball because it had become painful. The right eye had remained as when first seen in April, 1912. The enucleation was done September 10, 1913. Quite a large, dark, staphylomatous mass was found protruding down and backward from the region of the equator of the globe. The eyeball with the projecting mass was carefully removed and the specimen sent to the pathologic laboratory, where it was examined by Dr. Haythorn, the pathologist, and pronounced a melanosarcoma.

The orbit healed promptly. The patient was told of the diagnosis and probable prognosis, and that very likely there would be a return of the neoplasm. As he was very desirous of wearing a prosthesis, permission was granted, with a full explanation that the presence of the foreign body might act as a source of irritation. He was also cautioned that, if at any time pain or discomfort developed or if the orbital contents seemed to increase, or if the prosthesis should appear to bulge forward, to report at once, as this would in all probability mean a return of the neoplasm.

He wore the prosthesis with comfort and satisfaction until March, 1914, or about six months after the enucleation, when he returned. At this time he presented the appearance shown in Fig. 1. The orbital contents had decidedly increased. The prosthesis was pushed forward and downward, with a distinct bulging of the upper and lower lids. Palpation revealed the orbit to be quite well filled with a spongy mass limited only by the bony walls of the orbit.

Previous experience with orbital exenterations for malignant disease of the orbit decided the writer to try radium in this case, and on March 27, 1914, a deep incision was made into the orbit, practically bisecting it in its horizontal meridian, and 53 mgm. of radium element, in a silver capsule, screened by 2 mm. of lead and covered with a rubber tube, inserted into the orbit in its anteroposterior diameter, and held in place by means of a light compress and bandage and left for eleven and one-half hours.

The presence of the radium with its screening was somewhat painful, but did not cause any great discomfort. There was no febrile reaction. On April 13th, or seventeen days after the exposure to the radium rays, a distinct mass

could be palpated in the center of the orbit. This mass was dissected out on the following day, was 3 x 2.5 x 2 cm. in size, and proved to be strongly pigmented. This was sent to the laboratory, and the same diagnosis was made as of the enucleated eye, melanosarcoma. The orbit was lightly packed, and two days later 53 mgm. of radium element, screened as before, was inserted and allowed to remain in place for thirteen and one-half hours. The orbit did nicely, with a slight sloughing discharge, and was kept clean and healed promptly. It showed no evidence of any return of the neoplasm; at least there was no increase in the orbital contents, and after a few months presented the appearance shown in Fig. 2.

In November, 1914, the orbit became somewhat painful, especially at night, and there was a slight increase in its contents, especially along the nasal side. Palpation revealed a soft mass.

On December 7, 1914, quite a bit of strongly pigmented material was removed by curetting, and four days later 53 mgm. of radium element, screened as before, was inserted and left in place for twelve hours. This, as before, was followed by some sloughing from the orbit. The patient did nicely for six months, or until June, 1915, when the orbit again became painful and palpation revealed a movable mass within the orbit. On June 11th the orbit was again curetted, and 100 mgm. of radium element, screened as before, was inserted and left in place for fourteen hours. This, as before, was followed by sloughing, but the orbit continued to do nicely, and, when the patient was seen on July 12th, he complained of nothing except a burning sensation which he had not observed before.

He was not seen again until December 22, 1915, when the socket was partly filled, clean, and quiet, and the surrounding structures quite normal, as shown in Fig. 3. This photograph is slightly misleading, as in reality there was quite a depression, while the photograph makes the orbit appear quite full. The patient, however, appears pale and anemic. He stated that he had lost 25 pounds in weight, but felt quite well. There was no pain nor discomfort about the orbit. His blood was examined at this time by Dr. Haythorn, who reported: Red cell count, 4,716,000; leukocyte

count, 6,360; hemoglobin, 80 per cent. Differential count: neutrophiles, 63.5 per cent.; eosinophiles, 4.5 per cent.; basophiles, 1 per cent.; small mononuclears, 9 per cent.; large mononuclears, 12.5 per cent.; transitionals, 1.5 per cent.; unclassified, 8 per cent.

The patient was seen last on April 25, 1916. The orbit has remained quiet; no pain or discomfort and no increase in its contents. His general health, however, has been rather poor; he has lost some weight and was confined to his bed during the entire month of February. He now weighs 114 pounds, his normal weight being 150 pounds. His physician, Dr. Midgley, under date of May 5th, tells me that "there is a very extensive involvement of the liver, spleen, and pleura and probably of the bowels."

The value of radium, if used in sufficient amount, in malignant disease of the orbit is, in the opinion of the writer, clearly demonstrated by this case. The radium kept this neoplasm of the orbit in absolute abeyance, and the patient in a condition to follow his vocation up to within the last three months, or during a period of two and one-third years. The experience of this case has also suggested to the writer the use of the electric cautery knife, at a cherry-red heat, so as not to carbonize the tissues, for the removal of the malignant mass from the orbit instead of the knife, as has been suggested by others for the removal of malignant tissue from other parts of the body, so that when a similar case presents itself the writer will pursue the following technic: Make an incision through the skin of the lids, about 5 mm. from the free border of lids, completely encircling the palpebral fissure; then dissect the skin only of the lids back to the bony orbital wall; then with an electric cautery knife, at a cherry-red heat, by repeated punctures directed toward the apex of the orbit, and as closely to the bony wall as possible, completely sever the cone-shaped orbital content, and then introduce 100 mgm. of radium element and allow it to remain in place twelve hours, the radium to be introduced

again at some future time if necessary or advisable. The writer is strongly convinced that one application of 100 mgm. of radium element is of more value than several applications of a less amount.

The writer is indebted and hereby expresses his appreciation and gratitude to Dr. Wm. H. Cameron, of Pittsburgh, for the generous loan of the radium used in this case.

DISCUSSION.

DR. WM. ZENTMAYER, Philadelphia: I should like to mention a case in which the radium treatment proved far from satisfactory. It was a case of sarcoma of the orbit in a colored boy, ten years of age. Last fall I did a total exenteration of the orbit, including the periosteum. This was followed by the application of radium by Dr. Newcomet, who, I am sure, used the very best method under the circumstances. In three months' time there was recurrence in the upper-inner part of the orbit. In order to remove this, and to enable me to make a thorough examination of the orbit, I reflected the lids and removed all of the growth and all tissue still present. The patient was again referred to Dr. Newcomet, and the radium treatment continued, but after a lapse of three months a small indurated mass appeared behind the supraorbital margin and gradually increased in size until it protruded from the palpebral fissure. The patient was operated upon recently, and died about one hour after the operation was completed.

DR. W. E. LAMBERT, New York: I have had a similar experience in a case as just described by Dr. Zentmayer. The patient had a sarcoma at the inner canthus which was removed. The growth recurred in a few months, and was again operated upon. It again recurred, and the patient was referred to Dr. Abbe, who treated it with radium. Finally Dr. Abbe concluded to do a radical operation. The patient died on the table.

EXHIBITION OF NEW INSTRUMENTS AND APPARATUS.

A Practical Lamp for Ophthalmic Operations.

A. E. EWING, M.D.,
St. Louis, Mo.

The annoyance from poor illumination in searching for foreign bodies on the cornea and in delicate ophthalmic operations has prompted me to have constructed a lamp (Fig. 1) which has proved to be very satisfactory, especially because it is usually at hand, and may be readily applied to any electric light socket. It consists of an ordinary hand

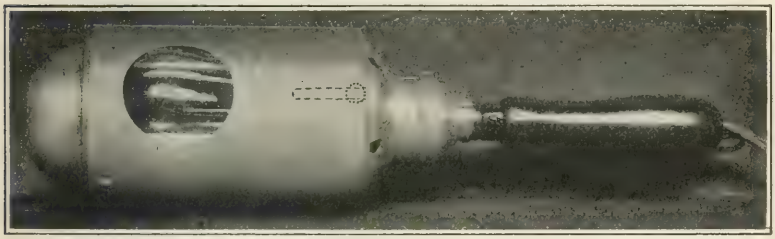


Fig. 1.—A practical lamp for ophthalmic operations. The slot and set-screw, which are on the opposite side to the light-opening in the hood, are indicated by dotted lines.

lamp, such as is used in workshops and garages, but the wire basket protector is replaced by a shade-holder and hood. To the circumference of the shade-holder a thin ring is attached, armed at one side with a set-screw, which plays in a slot in the open end of the hood into which the shade-holder slides, the other end of the hood being closed and the

opening for the light from the lamp being on the side near the closed end.

To insert the lamp the hood is removed, and when replaced may be adjusted by means of the set-screw to a 60 watt nitrogen or Mazda, or to a 15 watt Mazda or incandescent lamp. Both the closed end of the hood and the shade-holder contain ventilating spaces to prevent the overheating of the lamp, and they are so arranged as to intercept the direct passage of light. The diameter of the hood is 3½ inches; its length is 5 inches, not including the convexity of the closed end; the opening for the light is 2 inches in diameter; the total length of the lamp including the handle is 13 inches, and its weight with a nitrogen bulb is 18 ounces.

DISCUSSION.

DR. E. E. HOLT, Portland, Me.: In having a lamp holder made something like that exhibited by Dr. Ewing I found it to be much better when made of gun-metal, so as to avoid reflections from its surface. I do not see why these instruments should be made of brass or nickel, as when so made we are annoyed by the reflected light from the surface when using them.

A UNIFORM VISUAL TEST-OBJECT.

A. E. EWING, M.D.,
St. Louis, Mo.

The test-object here presented is offered as a compromise between the three line five and one minute test of Snellen and the broken ring test of Landolt, which was adopted as the International standard in 1909 by the International Ophthalmic Congress. The test of Snellen consists of a combination of objects, each subtending one and five minute angles, seen as one object subtending a five minute angle with the center of the object as the visual central

point. The Landolt broken ring consists of an object in the form of a ring, which subtends an angle of five minutes, the width of the line forming the ring subtending an angle of one minute, and at some point in the line there is a break, or an interval, which subtends an angle of one minute. Determining the position of this one minute break in the ring constitutes the test. This demands that the central visual axis be directed to the side of the object as a whole, instead

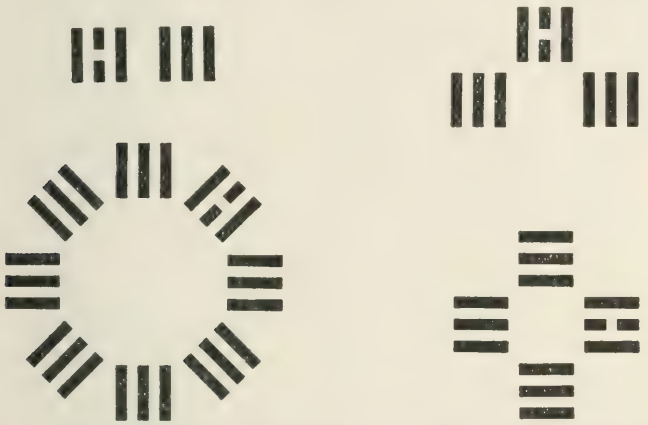


Fig. 1.—A uniform visual test-object illustrated in connection with the manner in which it may be grouped with the standard test-object of Snellen. Each figure in each group subtends an angle of five minutes at the standard five meter distance, and the width of each line subtends an angle of one minute.

of to its center, and the result is that there is greater irradiation or overlapping because the retina is stimulated on one side of the central visual point, and does not receive a similar stimulus on the other side.

The compromise here suggested combines the one minute interval in the Landolt test with the three line test of Snellen by placing the one minute interval in the center of the middle line of the Snellen standard. In this way an object is formed that contains the necessary requirements for

a one and five minute test-object, with the proper retinal stimulus on every side of the central one minute test.

To make this test-object available for practical application it is best to combine it with the three line test of Snellen in groups of two, three, four, or more, as indicated in the accompanying diagram (Fig. 1), and it is read in the same manner as the **E** of Snellen, or the broken ring of Landolt, by indicating its position in the group. In its employment with children it may be given a special name, as the "joker," the "little scout," or any term that will excite the interest of the child.

A CASE OF SYPHILITIC RETINOCHOROIDITIS JUXTAPAPILLARIS, WITH MICROSCOPIC EXAMINATION.

F. H. VERHOEFF, M.D.,
Boston, Mass.
(From the Massachusetts Charitable Eye and Ear Infirmary.)

The term retinochoroiditis juxtapapillaris is certainly descriptive of the pathologic condition found in the following case, although from an etiologic standpoint the latter probably does not belong to the class of cases described by Jensen.

James S., a negro sleeping-car porter, aged twenty-eight years, was admitted to the Massachusetts Charitable Eye and Ear Infirmary, May 29, 1915. He stated that one week previous, on arising in the morning, he found that everything appeared misty when seen with the right eye. Two days later the eye became congested and painful and the sight much worse. No previous eye trouble. (Later patient stated that he struck his eye on a door-knob on May 18th.) Patient denied venereal disease. (Later he admitted syphilis of tonsils, as noted below.) His general health was excellent.

Examination: Marked pericorneal congestion. Globe tender to touch. Cornea clear. Pupil semi-dilated (atropin), regular. Anterior chamber normal in depth, clear. Lens clear. Vitreous filled with dust-like opacities, preventing clear view of fundus. Retina can be seen as an indistinct, grayish-white membrane everywhere separated. Tension normal. Visual acuity of right eye, light perception only, projection faulty. Left eye normal; visual acuity, 20/20.

Treatment: Atropin sulphate, 1 per cent., three times daily. Mercurial inunctions twice daily. Aspirin. Hot applications.

June 2d. Morphin required for pain.

June 4th. Wassermann test reported negative.

June 10th. Small hypopyon. Pain less.

June 13th. Hypopyon gone. Posterior synechiæ are now present, although the pupil is still well dilated. No light perception.

June 16th. Vitreous very cloudy. Fundus not visible. Patient discharged against advice. Mercurial inunctions continued.

June 22d. Readmitted. Eye markedly congested. Cornea slightly hazy. Pupil irregularly contracted.

June 23d. Enucleation by Dr. Halliday.

June 28th. Patient stated for the first time that some months ago he was told at the Massachusetts General Hospital that he had syphilis of the tonsils, and that he was treated with salvarsan. From this hospital the following information has since been obtained in regard to the case:

The patient was first seen at the Massachusetts General Hospital February 25, 1915, when he complained of sore throat and swollen glands at the angle of the jaw. *Diagnosis:* Primary syphilis of left tonsil of one month's duration. No skin eruption. The patient was given 0.6 gm. of neosalvarsan and placed on mercurial treatment.

March 10th. Left tonsil still shows ulceration. Glands subsiding.

March 26th. 0.4 gm. neosalvarsan given. Mercurial treatment continued.

May 29th to June 28th, under mercurial treatment at Massachusetts Charitable Eye and Ear Infirmary (as noted above).

July 23d. Posterior surface of right thigh shows typical circinate lesion. Scattered papules on penis and scrotum. Mercurial treatment continued.

August 9th. Small patch on tip of tongue.

August 31st. Lesion on lower lip.

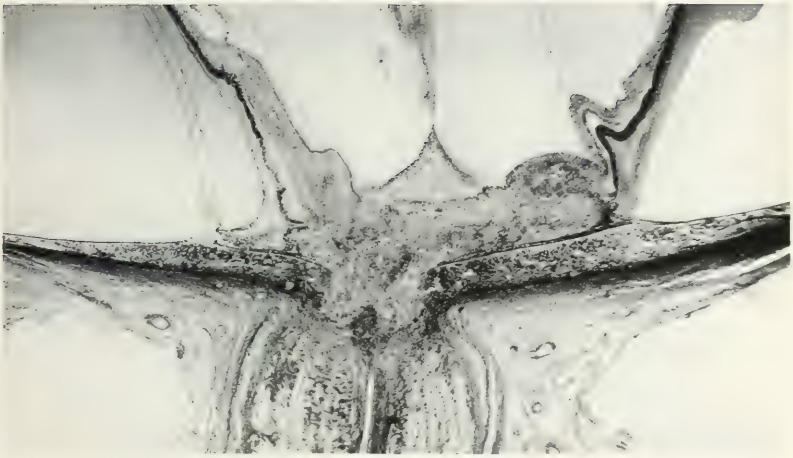
October 16th. Lesion on lip gone, but patches on tongue, tonsils, and anterior pillars persist. Salvarsan given.

November 19, 1915. No signs of active process in mouth.

Pathologic Examination (Path. Rec. 3–2788): The globe is normal in size and shape and the cornea is clear. The anterior chamber is normal in depth and filled with serum. The lens is *in situ*. The pupil, 3 mm. wide, is blocked by delicate membrane. The root of the iris is adherent to the ligamentum pectinatum for about 1 mm. all around. The vitreous is coagulated, after fixation, into a firm, semi-opaque mass. Near the posterior pole of the eye the retina shows a localized oval area of thickening about 7 mm. in diameter, which includes the disc and extends about 1 mm. beyond it. The center of the lesion is thus not in the disc, but in the retina above and to the outer side of the disc, where no doubt the lesion originated. In the affected area the retina in places reaches a thickness of 2 mm. and is adherent to the choroid. Everywhere else the retina is separated 1 mm. to 5 mm. from the choroid by serum, and shows numerous hemorrhages. On the temporal side the outer surface of the retina is coated with a thick layer of blood. The choroid beneath the retinal lesion is 1.5 mm. thick and gradually returns to its normal thickness within an area 16 mm. in diameter.

Histologic Examination: Fixation for forty-eight hours in an aqueous solution containing formalin, 1 per cent., alcohol, 52 per cent., and phenol, 5 per cent. Part of the affected retinal lesion prepared by the Levaditi method. Horizontal sections through the middle of the retinal lesion and optic disc. Staining by a variety of methods.

The chief pathologic process in the eye has to do with the localized lesion involving the retina and disc. Within the area of this lesion the normal structures have been almost completely replaced by granulation tissue richly infiltrated with chronic inflammatory cells. The granulation tissue extends just through the lamina cribrosa, but does



Syphilitic retinochoroiditis juxtapapillaris (× 8).

not invade the nerve stem. The cellular infiltration consists mainly of plasma cells, but lymphocytes are also abundant; only a few eosinophiles and pus cells are present. There is marked venous congestion, and here and there interstitial hemorrhages and blood-pigment are seen. The inner surfaces of the retina and disc are in most places intact, but just to one side of the disc the surface of the retina has been broken through by an exuberant growth of granulation tissue which is pushing its way into the vitreous. This is coated with blood and fibrin.

The blood-vessels in the disc and affected portion of the retina show marked proliferative endovasculitis, especially the arteries. A main branch of the central artery is almost completely obstructed by endarteritis and farther on is thrombosed. Many of the smaller vessels contain fibrinous thrombi, some of which are infiltrated with necrotic cells. Still other thrombi contain hematogenous pigment and show advanced organization.

The retina away from the lesion proper is everywhere more or less necrotic and shows numerous hemorrhages, some of which extend into the vitreous or more often into the subretinal space. The outer surface of the retina is coated with fibrin.

The nerve stem shows marked venous congestion, interstitial hemorrhages, and dense perivascular infiltration with lymphocytes and plasma cells. The central vessels up to the lamina cribrosa are almost free from endovasculitis. The artery, however, shows here and there a few fatty endothelial cells in the intima.

The choroid beneath the retinal lesion is highly congested and greatly thickened by edema and infiltration with plasma cells, but it has not been invaded by granulation tissue. The pigment epithelium has been separated from the membrane of Bruch by a layer of plasma cells, and is partly disintegrated. The membrane of Bruch, however, is for the most part intact, but in a few places has been broken through. The infiltration of the choroid rapidly diminishes and entirely ceases at a distance of about 1 cm. from the disc all around.

The vitreous body is infiltrated with serum throughout. Anteriorly it is relatively clear, containing only a few

lymphocytes and a considerable amount of fibrin evidently given off at the ora serrata. Posteriorly, it contains masses of necrotic cells apparently derived from the retina, much fibrin, and many fresh pus cells.

The cornea is normal. The tissue of the limbus is congested and moderately infiltrated with lymphoid cells. On the posterior surface of the cornea there are occasional single lymphoid cells but no clumps of cells. There is no trace of hypopyon.

The root of the iris is firmly united with the ligamentum pectinatum. The anterior surface of the iris is coated with a thick vascularized membrane. This does not intervene between the root of the iris and the ligament, but continues over the lens surface. The pupillary margin of the iris is united to the lens. The iris stroma is congested, but not swollen, and contains only a very moderate number of chronic inflammatory cells. The lens is normal.

The ciliary processes are giving rise to a slight exudation of lymphoid and pus cells, but otherwise the corona ciliaris is almost normal. The orbiculus ciliaris, however, is markedly altered. It is greatly infiltrated with lymphoid cells, and its epithelium is being disintegrated by granulation tissue springing from its surface. The granulation tissue is thickest at the ora serrata. Here its free surface is richly vascularized and it is actively proliferating into the vitreous, but it does not appreciably involve the choroid.

Bacteriologic Examination: Sections stained by the Levaditi method, by the Gram method, in carbolthionin, and for tubercle bacilli, are all negative for microörganisms. (It is probable that the fixative employed was not suitable for the Levaditi method.)

The important features of this case may be briefly summarized as follows:

A man, aged twenty-eight years, developed primary syphilis of the left tonsil and was treated at a general hospital with neosalvarsan and mercury for about one month. At the end of this time the tonsil was much improved and the patient discontinued treatment. About two months later the patient for the first time noticed that the sight of one eye

was blurred, and one week after this the eye became painful, and there were found slight iritis, abundant fine vitreous opacities, and separation of the retina. A Wassermann test was negative. In spite of mercurial inunctions twice daily for three months the inflammatory condition of the eye and the pain continued to increase and the eye became totally blind and was removed. On pathologic examination there was found a granulomatous lesion centered near the margin of the disc which involved chiefly the inner layers of the retina and only secondarily the disc and choroid. About three weeks later definite secondary syphilitic lesions of the skin and mouth had developed. After a further course of mercurial treatment for four months all the lesions had healed.

This case in many respects was not unlike a case of syphiloma of the optic nerve and papilla previously recorded by me, in which there were likewise marked inflammation of the anterior part of the eye and great pain. The lesion, however, was centered in the nerve stem and involved the disc and retina secondarily. In this previous case I was fortunate enough to be able to demonstrate an abundance of spirochætes in the lesion, and it seems to be the only case of its kind in which these organisms have been demonstrated.

In the present case, the history of contusion of the eye, which was given as an afterthought by the patient, is of questionable significance, although it seems possible that the blow may have determined the localization of the retinal lesion.

Retinochoroiditis juxtapapillaris was first described as a clinical entity in 1909 by Jensen, who reported four cases. According to his description the affection occurs in healthy young persons without evidences of syphilis or other constitutional disease. Ophthalmoscopically there is seen a light prominent infiltrate immediately beside the papilla, oval in form, and about the size of the papilla. The retinal vessels within it are partly or wholly obscured. The choroid is also affected, as evidenced by vitreous opacities. The rest of the

fundus is normal. The subjective symptoms consist in the blurring of vision due to the vitreous haze, and in a sector-like defect in the field extending from the blind spot to the periphery. In the course of a few months the infiltrate begins to absorb from its periphery, finally leaving a pigmented white spot, as in the case of any retinochoroiditis. The vitreous opacities also disappear. After several years recurrences may take place in the same area. In none of Jensen's cases was central vision affected.

Since Jensen's report, nineteen other cases have been recorded in the literature, but no further light has been thrown on the etiology. Jensen contended that the scotoma was due to the cutting off of the blood supply of the affected area. Groes-Petersen, however, and subsequent observers, give good reasons for believing that it is really due to destruction of the nerve-fibers in the retinal infiltrate. Petersens also described two cases in which pigmentary changes were present in the macula of the opposite eyes, and van der Hoeve has recently reported a case in which such changes were present in the macula of the same eye, and pointed out that in six of the twenty-five eyes of the reported cases central vision was permanently impaired. He also combined all the fields of these eyes and found that the upper-outer quadrant always escaped. Still more recently Rönne has reported two cases in each of which the sector defect in the field involved the upper-outer quadrant. In one of these cases there were two fresh infiltrates away from the macula and disc, and old macular changes in the other eye. In the other case there were precipitates on the back of the cornea. Thus far no eye affected with this disease has been examined microscopically.

It will be seen that the present case was similar to Jensen's cases in the situation of the retinal lesion, in the fact that the latter was solitary and affected most severely the nerve-fiber layer, and in the occurrence of vitreous opacities. It differed

from his cases in the far greater intensity of the process, in the occurrence of inflammatory changes in the anterior part of the eye, and in the existence of positive evidences of syphilis. It is evident, however, that if the case had been of a milder type and the retinal lesions had ultimately healed, the condition probably could not have been distinguished from Jensen's retinitis. The iritis and other inflammatory changes in the anterior part of the eye were clearly due to toxins diffused through the vitreous from the retinal lesion, and in a mild case would not have occurred.

It therefore seems possible that some of the cases reported as instances of Jensen's retinitis were syphilitic in origin. In favor of this assumption also is the tendency of Jensen's retinitis to recur in the same place. That the majority of cases of this kind, however, are syphilitic in origin is improbable. More likely they are due to an unknown infectious agent which tends to produce single foci in the choroid or retina. When one of these foci occurs in the retina at the disc margin, the condition is classified as Jensen's retinitis. When it occurs elsewhere, as probably it more often does, the condition is regarded simply as chorioretinitis due to some unknown cause.

REFERENCES.

1. Edmund Jensen: "Retinochoroiditis juxtapapillaris," Arch. f. Oph., 1909, lxix, p. 41.
2. Groes-Petersen: "Retinochoroiditis" (Edmund Jensen), Klin. Monatsbl. f. Augenh., 1912, ii, p. 159.
3. J. van der Hoeve: "Nervenfaserdefekte bei Retinochoroiditis juxtapapillaris," Klin. Monatsbl. f. Augenh., 1914, liii, p. 487.
4. Ronne: "Ueber die Retinochoroiditis," Klin. Monatsbl. f. Augenh., 1915, liv, p. 455.
5. Verhoeff: "Ein Fall von Syphilom des Optikus und der Papille mit Spirochätenbefund," Klin. Monatsbl. f. Augenh., 1910, xviii, p. 315.

DISCUSSION.

DR. T. B. HOLLOWAY, Philadelphia: I would like to ask Dr. Verhoeff whether his studies would permit him to state whether this exudate or granulomatous tissue might exist

without being severe enough to cause this sector-like defect in the field? If I mistake not, Groes-Petersen alludes to several cases where this field defect was absent. I mention this because at the present time two patients are under observation on the service of Dr. de Schweinitz at the University Hospital who present typical exudates adjacent to the disc, and in each instance there is a well-defined patch of exudative choroiditis down and in in the mid-zone. As far as the lesions are concerned, the cases are identical; one had a sector-like defect and in the other this was absent. In both instances the Wassermann test was negative, yet in one improvement under inunctions was more rapid than is usually seen in other than specific cases. In the other case the clinical findings at the first examination were negative aside from a distinct anemia. A third case has only recently come under observation, and in this patient there is no peripheral lesion and the exudate near the disc is much less pronounced. As yet she has not been sufficiently studied to permit a definite report to be made. An interesting clinical fact connected with the patient with the anemia is that she is the sister of a syphilitic patient that I referred to in a discussion before this Society two years ago, who had visible blood streaming. The mother of these two patients is now under treatment at the dispensary for an extensive uveitis. Since these remarks were made she has died as a result of an apoplexy.

Dr. Wm. Zentmayer, Philadelphia: Soon after Jensen published his article upon this interesting condition I reported a typical case and called attention to the fact that in order to make a positive diagnosis there should be a typical field defect, and, if a recurrence of the choroidal lesion took place, it should be at the same locality, otherwise an ordinary retinochoroiditis with the accidental location of the lesion in juxtaposition with the papilla might be mistaken for this condition.

Dr. G. E. de Schweinitz, Philadelphia: Recently I have had under observation a girl, nineteen years of age, who presented the classic ophthalmoscopic appearances of retinochoroiditis juxtapapillaris, and with the characteristic visual field defect. The patient was most thoroughly examined

by various internists and special laboratory assistants. The only abnormal condition which was found was a persistent simple anemia. When this was brought under control and a faulty diet corrected, there was rapid subsidence of the lesion, and there is now restoration to practically full visual acuteness. In another somewhat similar case, although perhaps not quite so typical, a like condition of affairs was present, with an equally happy result.

DR. F. H. VERHOEFF, Boston, Mass.: One observer I think has reported some cases in which the scotoma was only relative, not absolute; but I do not believe any one has reported a case without scotoma. I have seen two clinical cases, such as Dr. Holloway reported, in the past year, but I am in doubt whether to classify them as Jensen's retinitis or not. In these cases the exudates seemed to come out of the porus instead of starting at the edge of the disc. These cases had no sector scotomata at all, and had some foci around the macula, such as some other observers have described. Both patients gave negative Wassermann tests, and both gradually improved, one under mercurial treatment and the other without any medicinal treatment.

TUBERCULOSIS OF THE RETINAL VESSELS.

EDWARD JACKSON, M.D.,
Denver, Colorado.

For the practical management of cases, the diagnosis of intraocular tuberculosis must rest on the clinical picture revealed by the ophthalmoscope and the reactions to specific tests. Studies in pathologic anatomy can give a definite basis and significance to the ophthalmoscopic findings, but they are not available when the case presents for prognosis and treatment. Practical advances in regard to these conditions must largely depend on a definite and complete appreciation of the ophthalmoscopic symptoms to which they give rise.

Tuberculosis starts as a local disease, and, unlike syphilis, as regards anatomic lesions it probably remains a local disease in many cases. Some of the general symptoms commonly associated with it arise from mixed infection and the general effects of pulmonary involvement. The earliest known and most common general manifestation of the disease is that systemic resistance manifest in the tuberculin reactions. Even this is not constant, or it is concealed by unknown conditions, as we have learned by failures of the various tuberculin tests. Probably with primary intraocular tuberculosis the conditions surrounding the local lesions especially oppose the development of any general manifestations whatever; von Hippel reported a case of tuberculosis in which a febrile reaction was obtained only from the sixth injection of old tuberculin in dose of 5 mgm. I have seen a case of intraocular tuberculosis in which no reaction was obtained from a first diagnostic injection of tuberculin; and in a case of general bilateral corneoscleral-uveal tuberculosis, with subsequent tuberculous meningitis, the von Pirquet test proved completely negative. In the case on which this paper is largely based the von Pirquet test proved negative, although an injection of tuberculin gave systemic and focal reactions. This does not show that the tuberculin diagnostic tests are not of great value, but it does emphasize the importance of getting as correct and definite a conception as possible of the ophthalmoscopic symptoms, an importance that justifies the presentation of the accompanying sketches of them.

Within the last two years the writer has seen five cases presenting the ophthalmoscopic appearances of retinal tuberculosis. Three of these, in which the diagnosis was fully supported by the results of tuberculin injections, were under regular observation for many months. Two of these have been reported last year (Trans. First Colorado Ophthalmological Congress). The case on which the accompanying

Tuberculosis of Retinal Vessels.
Upper—Early stage. Lower—Late stage.

plate is based has been under observation for over eighteen months; and throughout nearly the whole period presented conditions exceptionally favorable for ophthalmoscopic study. Vision in the one eye affected was at one time reduced to counting fingers at six inches, but has risen again to 0.8, and the eye has remained without fresh lesions about a year. The title tuberculosis of the retinal vessels has been used because, in these cases, the symptoms and visible lesions are strikingly connected with the blood-vessels.

In all these cases there was localized clouding of the vitreous not definitely connected with any retinal lesion. In the case here represented there were these vitreous clouds with indefinite edges and dense centers that were not connected with any hemorrhage or visible vascular lesion. These were well inside the vitreous, and seemed quite surrounded by relatively clear vitreous. In every case there was also from time to time diffuse clouding of the vitreous from hemorrhage. But in the case here represented the general clouding occurred but once, near the beginning of the disease.

In every case there were hemorrhages. In the one here illustrated these hemorrhages, except the first, were small and confined to the retina. It was this feature that made the case so favorable for ophthalmoscopic examination. In all the other cases the hemorrhages repeatedly broke into the vitreous, giving the typical picture of recurring retinal hemorrhage in young persons.

A most striking ophthalmoscopic appearance in the case here represented is that of the rounded spots, smaller than the optic disc, with indefinite margins, at first light gray in color, as the one above the disc, later becoming vascular, as the one to the nasal side of the disc, always located on one of the larger vessels and finally disappearing, to leave the region involved almost or quite normal. In the beginning the patch had some resemblance to a choroidal tubercle; but was whiter, less yellow in color. The mass completely con-

cealed the large vessel that passed through it. In no case did any alteration of pigmentation follow. The impression gained was that each of these spots represented an exudate involving the vessel-wall and the adjoining vitreous—a lesion that might be provoked by the presence of a colony of tubercle bacilli, but which probably would not exhibit the structure of a fully formed miliary tubercle.

The most obvious alteration in any of the vessels was exhibited by the upper temporal vein. Toward the periphery this appeared normal in color and contour, or slightly dilated, but at a certain point it abruptly widened out to full four times its normal diameter, and became gray, tinged with the color of the normal venous blood-column. Its sides were nearly parallel and comparatively straight. It appeared to be a swelling of the vessel-wall, with dilatation of the vessel lumen. This condition changed slowly during five or six months, and returned gradually to the normal. This lesion had scarcely begun to undergo involution when the rounded spots above referred to, and the white dots next to be mentioned, had almost entirely disappeared.

An interesting part of the ophthalmoscopic picture is the small white spots in the macula. These were generally less brilliant than the spots of renal retinitis or papilledema, although they approached that appearance. The largest were two or three times the diameter of the largest normal retinal vessels, and their edges faded gradually into the normal fundus surrounding them. They developed gradually and faded away gradually, some more rapidly than others, but all were evolving or undergoing retrogression together. They appeared to be the manifestations of some general condition affecting the nutrition of all that part of the retina, and not separate centers of pathologic activity arising independently of each other, like local infections. Spots of this kind were noticed in two of the five cases, and they have been noted by other observers, so that they must

be regarded as an important ophthalmoscopic symptom of this disease. Knapp* recorded them in one of his cases, although the radiating figure shown in his plate bears more resemblance to the star of renal retinitis than did the grouping of these spots seen in my patients. These spots disappeared entirely about the time that central vision had risen to 0.7. They left no trace of altered pigmentation.

About the time the diagnostic injection of tuberculin was given in the case represented, and in one of the other cases while under observation, the optic disc became hidden in a gray, non-vascular haze with an indefinite edge, that a little later became vascular. The appearance came to resemble that of an increasing papilledema, except that the central retinal vessels were not choked and the boundaries of the swelling were less abrupt. This swelling showed no tendency to give place to post-neuritic atrophy, but gave rise to a permanent condition of white connective tissue, poorly supplied with vessels, but sometimes showing on its surface large new-formed vessels typical of retinitis proliferans. In the case shown this scar tissue reached forward 5 D. in front of the disc. The strands extending toward the lower periphery of the fundus were less prominent. In other cases the scar-tissue was much more extensive, stretching forward to the extreme periphery of the fundus, and from 10 to 20 D. in advance of the normal surface of the retina.

These lesions showed marked focal reactions to tuberculin injections. In the case shown the swelling over the nerve-head became highly vascular, changed color completely within forty-eight hours after the diagnostic injection that raised the body temperature one degree, and minute hemorrhages occurred after other injections. In another case on two occasions within forty-eight hours after therapeutic injections large vitreous hemorrhages occurred, greatly reducing the vision that had been regained.

* Trans. Amer. Oph. Soc., xiii, p. 486.

Another patient without active lesions for over nine months had vision slowly improving up to 0.4. She had become pregnant, went to term, bore a healthy child, and nursed it. But when the child was three months old the trouble recurred, and a hemorrhage reduced vision in one eye to moving objects.

None of these patients has shown marked evidence of pulmonary tuberculosis, although I have seen a case in which the ocular lesions appeared late in that disease. But all of them showed some moderate anemia and some departure from robust health.

The clinical picture that it seems important to fix in relation to retinal tuberculosis includes these features: vitreous opacities, recurring hemorrhages, enlargement of the retinal veins, the local lesions described associated with large retinal vessels, white spots in the macula in some cases, optic neuritis, and retinitis proliferans as a terminal condition; the earlier lesions giving reactions to tuberculin injections, and their involution favored by tuberculin therapy.

DISCUSSION.

DR. F. H. VERHOEFF, Boston, Mass.: I believe that Dr. Jackson did not say anything about the pathologic examinations that have been made in these cases. There seem to be two types of retinal hemorrhages due to tuberculosis, one occurring in miliary tuberculosis and the other in chronic cases. Stock has made a microscopic examination of one case of miliary tuberculosis and finds that you may get retinal hemorrhages with no tubercle formations in the retina. Possibly the bacilli have lodged there and caused hemorrhages before any tubercles have been formed. The more interesting cases, however, are those of the chronic type of which Dr. Jackson speaks, and which are practically never fatal. The patients are in fairly good health, and if they did not have the eye condition would not be suspected of having tuberculosis. Fleischer, in 1914, examined one of these cases microscopically, and found actual tubercles

around the retinal veins. He found also slight tuberculosis of the anterior part of the eye, and thought the retinal tuberculosis due to the bacilli getting into the retina anteriorly and being carried along the vessels, lodging at different places and causing tubercles and hemorrhages. If his view is correct, it falls in line with the view that I have advanced as to the origin of chronic ocular tuberculosis in general; namely, that it is due to the secretion of the bacilli into the aqueous from the ciliary processes. In one case of marked tuberculosis of the ciliary body that I examined microscopically I found tubercles in the retina, such as Fleischer described. I think it is still debatable as to whether Fleischer is right in claiming that tuberculosis of the retina is ordinarily produced in this manner, but it would certainly simplify the whole question if we could show that all forms of chronic tuberculosis occurred in the same way.

DR. W. B. WEIDLER, New York: May I ask Dr. Jackson to tell us something of the technic in the treatment of the cases with tuberculin?

DR. CARL FISHER, Rochester, Minn.: Referring to one of the cases Dr. Jackson mentions as giving the picture of recurrent retinal hemorrhages in a young person; I was on the program for a report of a similar case with an experimental study on animals, but certain late developments have rendered the experimental evidence not conclusive enough to warrant me in reporting it as a separate paper. I think, however, that the case may possibly come under this classification which Dr. Jackson mentioned. A young man began having hemorrhages at the age of nineteen, and I saw him when he was twenty-three years of age. The Wassermann, the complement-fixation test for gonorrhea, and every possible means of general diagnosis were tried, and everything was negative except the tuberculin reaction. This was always positive. That would make it look like a case of tuberculosis pretty definitely, but the interesting fact was that the patient did not get better under tuberculin therapy, and he was then given salvarsan elsewhere, which resulted in the permanent loss of vision in the left eye. When I first examined this eye there was a white mass that covered the blood-vessels and looked like suppuration of the retina.

About a year later I saw him, and he had a cloudy opacity of the vitreous in the right eye. In the course of examination pus was found coming from a molar tooth, and when the patient did not keep it clean by sucking, etc., the vision would go down. In order to see what would happen I asked him to leave the tooth alone for twenty-four hours, with the result that vision sank to shadows. This was repeated several times, and we concluded that it was quite probable that the focus of infection was the cause instead of tuberculosis. The tooth was extracted, and after that he had the longest period of freedom from hemorrhages that he had ever had. Dr. E. C. Rosenow made cultures from this pus, and we injected it into eight rabbits, and five developed little patches of exudate beneath the retina and in the choroid. The interesting point in the case to me was the marked improvement which occurred on getting rid of the infection and the absolutely bad course which the disease pursued under tuberculin therapy. I hope to be able to settle the question of etiology in this case definitely, for I believe that there is a strong probability that many at least of these cases are due to focal infections.

Dr. Arnold Knapp, New York: It is remarkable how frequent these cases of retinal tuberculosis are if one is on the lookout for them. It seems to me that we can classify them somewhat as follows: Aside from the form with large exudations, which corresponds to the Coats type of exudative retinitis, there is a variety in which there are small exudations and hemorrhages. This is a form of which Dr. Jackson's is an example, and which, in my experience, are unusually amenable to tuberculin therapy.

The third variety is the one which is characterized with hemorrhages which vary in extent. In some of these the veins show distinct perivascular exudations. In other cases, however, there is no lesion of the vein to be determined with the ophthalmoscope. During the past winter we have had an opportunity of following a case just like that reported by Dr. Fisher, in which retinal hemorrhages occurred in a young man who had a very definite abscess at the root of a tooth. The extraction of this tooth had, however, no effect on the retinal process. The patient gave a very distinct tuberculous history and reacted to tuberculin.

This etiology is still further borne out by a subsequent appearance of retinal hemorrhages in the other eye.

Dr. Jackson mentioned the spots seen in the macula, and said, if I understood him correctly, that they were the same as in albuminuric retinitis or in neuroretinitis. I have the impression that the spots in neuroretinitis are of a different type, and that those caused by retinal tuberculosis belong in this group and distinctly differ from those in albuminuric retinitis.

Dr. Edward Jackson, Denver, Colo.: With reference to the pathology of these cases, within the last eighteen months there has been published a paper by Dr. Otori describing retinal periphlebitis. But in the cases I reported, and especially in the one here illustrated, the majority of the lesions that looked most like tubercles were connected very distinctly with the larger arteries. It is quite possible that they arise by the migration of the bacillus from small lesions anteriorly; and the appearance of the vitreous haze, apparently isolated from any retinal lesion, would point in that direction. Some of these lesions, however, were in close relation with the arteries.

A CLINICAL AND PATHOLOGICAL STUDY OF TWO CASES OF MILIARY TUBERCLES OF THE CHOROID.

ROBERT L. RANDOLPH, M.D.,
Baltimore, Md.

AND

H. C. SCHMEISSER, M.D.,
Baltimore, Md.
(By invitation.)

(From the Departments of Pediatrics and of Pathology, Johns Hopkins University, Baltimore, Md.)

Jaeger,[1] in 1855, first described ophthalmoscopically a choroidal tubercle,* and his observations were confirmed by von Graefe and Leber.[2] The earlier ophthalmologists, how-

* Whether miliary, has since been questioned.

ever, insisted that ophthalmoscopic changes in tuberculous meningitis were rare, and it is with no little interest and surprise that one reads the comparatively recent observations of Carpenter and Stephenson,[3] who claimed to have found choroidal tubercles in 50 per cent. of their cases. Following close upon their communication we find the work of Marple[4] in the Babies' Hospital, N. Y., who concludes that in tuberculous meningitis choroidal miliary tubercles are the rule, (100 per cent. in 13 cases), a statement made at the Ophthalmological Congress in Oxford in 1912. And yet in contradiction to Marple we find Heinzel[5] with negative findings in 41 cases of tuberculous meningitis which he examined. We have then, in these latter days, in opposition to the rather conservative statements of the earlier ophthalmologists, the figures of Carpenter and Stephenson and Marple, who place the percentage at from 50 per cent. to 100 per cent. These more recent figures are based upon frequent examinations, which indicate how often the changes were overlooked in old times when one routine examination was made and as often nothing found. The eye-grounds were not subjected to further examination, and the case was recorded as negative so far as tubercles in the choroid were concerned. It should be remembered that the tubercles are usually seen among the terminal events in the history of the case, and ophthalmoscopic examinations should be frequently made. Marple mentions a case in which an ophthalmoscopic examination made at 6 P. M. disclosed fundi entirely free of tubercles, and at 10 P. M. a tubercle was found, this having developed in the interval. The introduction of the electric ophthalmoscope makes examinations of this character so much easier. Not only is the fundus perfectly seen, but the great difficulty in conducting successfully an examination of this character in the case of a baby is reduced to almost a minimum by having a brilliant, steady, and uniform illumination. It is certainly easy to

Fig. 1.—Case I. Eye-ground of right eye. × 5.

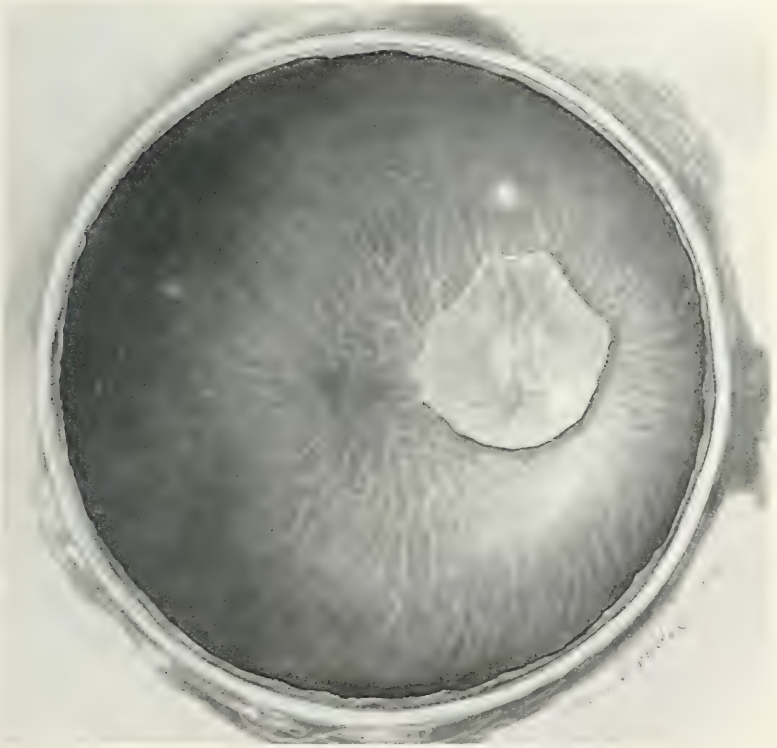


Fig. 2.—Case II. Eye-ground of right eye. × 5.

overlook changes of this nature when using an ordinary non-electric ophthalmoscope, an examination which often presents great difficulties.

In the first case, Dorothy Ziemen, the tubercles, three in number, were found in the right eye above the disc, and at an angle of 120 degrees from the vertical meridian, while in the left eye there was a tubercle above, one below, and one to the nasal side of the disc. In the second case, Dorothy Chapman, there was a tubercle in the right eye above the disc and one far out to the temporal side. What was seen in the left eye could not be regarded positively as a tubercle. It might be well to state here that it is important to make the examination in both eyes, for the tubercles often occur in one eye and not in the other. It is not necessary to describe them ophthalmoscopically, as their appearance is sufficiently familiar to us all.

What is particularly striking in connection with this subject is, first, the necessity of using the electric ophthalmoscope, and, second, the importance of frequent examinations —at least twice daily. A cycloplegic should always be used. These conclusions may be equally well applied to the examinations of the eye-grounds in cases of Bright's disease. Statistics based upon infrequent examinations may be regarded as more or less unreliable and as giving an inaccurate idea of the frequency of ophthalmoscopic changes in nephritis. The same rule might be adopted in not only Bright's disease, but in the various blood diseases, as, for instance, pernicious anemia. In both of these diseases we are in the habit of making a routine ophthalmoscopic examination when first seen. We are less apt to repeat such examination unless subjective symptoms, such as dimness of sight, should develop and call our attention to the possibility of ophthalmoscopic changes having suddenly appeared since the first examination.

CASE I.—Dorothy Ziemen, aged two years. The father died of tuberculosis, with hemorrhage, when patient was sixteen months old. Mother is living and in excellent health. She has never had any miscarriages. The child at birth weighed nine pounds and was fed at the breast until she was eighteen months old, when she began to eat at the table with the rest of the family. She has never been sick in bed, but had pertussis. She became sleepy and cross, and not long afterward began to vomit, and did this whenever she was moved. She had no appetite and was constipated. The temperature was 99.4° F., pulse 100, and respiration 24. She lay in bed and rarely moved unless disturbed, with lids half shut and mouth wide open, breathing slowly, with periods of apnea. She shunned the light and was very irritable. There was no general glandular enlargement. The neck was somewhat rigid. A few days later the spinal fluid showed a film on standing, and the eye-grounds began to show blurring of the edge of the disc; four days later tubercles were discovered in the eye-grounds.

CASE II.—Dorothy Chapman, aged four months. Born at full term, and had been free of illnesses until the present one. The onset was characterized by fever and cough. She cried a great deal and slept very little. The temperature was 102° F., and the respiration 84. She lay on the table with distant expression, hands tightly clenched, and legs flexed. The reflexes were slightly exaggerated, and there were numerous tuberculides all over the body. The tubercles were found in the choroid at the first examination.

PATHOLOGICAL EXAMINATION.

Autopsy of these two cases revealed the following:

CASE I.—General miliary tuberculosis with acute tuberculous meningitis. The portal of entry of the organism was most probably through the alimentary canal, where ulcers were found. The tubercles were widely scattered through the body, being present in the mesenteric and pulmonary lymph-glands, lungs, pleura, endocardium, spleen and its capsule, liver and its capsule, and last, and most important, in the choroid membrane of both eyes.

Fig. 3. Case I. Left eye. × 45. Below is the sclera and above the vascular and pigmented choroid. The tubercle is limited to the latter. It shows a typical giant-cell, epithelioid and small round cells, with the characteristic grouping.

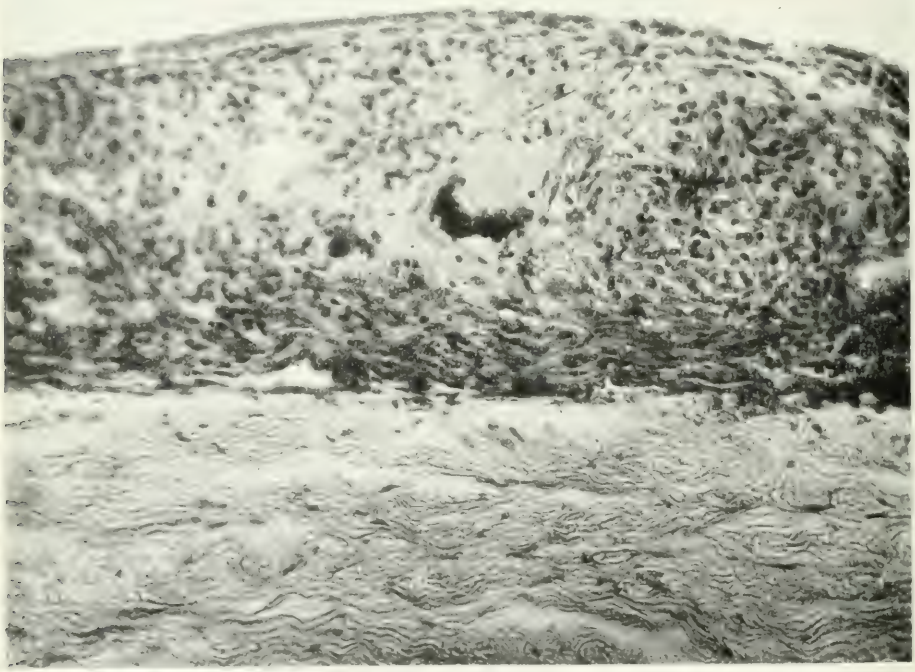


Fig. 4.—Same as Fig. 3, a higher power. × 152.

On opening the right eye (Fig. 1) there was nothing abnormal noticed until the retina was removed, then one could see a number of just visible, slightly larger than pin-point, bluish-white, translucent nodules projecting from the black surface of the choroid. Taking the macula lutea as the posterior pole, two of these miliary tubercles were located in the upper temporal quadrant and a third in the upper nasal quadrant. Several more were seen with a magnifying glass, but their exact position was not noted.

In the left eye, again after the removal of the retina, there were seen in the upper nasal quadrant two and in the lower temporal quadrant one miliary tubercle rising from the choroid. They were very similar in size, shape, and appearance to those of the right eye.

Of the left eye a careful microscopic examination was made (Figs. 3 and 4). A section taken perpendicular to the surface, through the choroid and sclera, to include one of the small bluish-white nodules, presented the following picture: To one side of the section was seen the thick sclera, composed of rather coarse strands of wavy fibrous tissue with scattered, deeply staining, spindle-shaped nuclei. Only one or two blood-vessels were present. Joining the sclera was the choroid, a membrane of only about one-tenth the thickness of the former, and composed of loose cellular connective tissue with large pale nuclei and many blood-vessels. The fine brown pigment granules were found principally in the cells along the inner margin and in a single row of cuboidal cells—the retained layer of pigment-cells of the retina. Limited to the choroid was a single miliary tubercle, showing the characteristic structure and constituent elements. In its center was a typical giant-cell, surrounded by epithelioid cells with a peripheral zone of lymphoid cells. There was no caseation.

Sections stained for tubercle bacilli showed the same to be present in moderate number within the tubercle.

Case II.—General miliary tuberculosis without meningitis. The portal of entry was not definitely determined, but was thought to be through the lungs. The tubercles were found widely disseminated through the body, even involving the skin. They were present in the choroid of the right eye, but could not be demonstrated in the left.

The retina of the right eye (Fig. 2) appeared uninvolved, and no tubercles could be seen through the same, but after removing the retina two small gray nodules were present in the choroid. In shape and appearance they were similar to those of the first case, except that they stood out in stronger relief from the somewhat darker choroid of the negro infant, but in size the one was almost 1 mm. in diameter, the other one just visible. The first was located in the upper nasal quadrant, slightly above the optic disc, and the second in the periphery of the upper temporal quadrant.

The literature of miliary tubercles of the choroid shows that the first case described both grossly and microscopically was by Manz[6] in 1858.* Five years later he[7] added two more cases. His findings were soon confirmed by Busch,[8] who contributed the fourth case. The following year Cohnheim[9] published seven cases that he had observed during the previous winter, thus taking this lesion out of the realm of pathological curiosities. Nevertheless, it can hardly be said that the literature is overrun with pathologic data on this subject. Isolated cases were reported until Litten,[10] in 1877, tells us that he found choroidal tubercles postmortem in 39 out of 52 cases of acute miliary tuberculosis. Since then the purely pathological literature has not been so abundant as might be expected.

Most of the cases of miliary tubercles of the choroid have occurred in cases of general miliary tuberculosis with or without tuberculous meningitis. However, it is well known that they may be found, but not so frequently, in practically any type of tuberculosis with a primary lesion almost anywhere in the body.

CONCLUSION.

The two cases of miliary tubercles of the choroid reported above are of interest because:

* Jaeger in 1855 examined the choroidal nodules in his cases anatomically, and pronounced them tubercles, but it has since been doubted whether they were miliary tubercles.

1. In the one case we are dealing with general miliary tuberculosis with acute tuberculous meningitis, and in the other with general miliary tuberculosis without meningitis.

2. The miliary choroidal tubercles were demonstrated during life, and verified at autopsy both grossly and microscopically, including the demonstration of the tubercle bacillus in the lesion.

BIBLIOGRAPHY.

1. Jaeger, E.: "Ueber Chorioidealtuberkel," Oesterr. Zeitschr. f. prakt. Heilk., Wien, 1855, i, 9–10.
2. V. Graefe, A., and Leber, Th.: "Ueber Aderhauttuberkeln," Arch. f. Ophth., Berlin, 1868, xiv, part 1, 183–206.
3. Carpenter, G., and Stephenson, S.: "Tuberculosis of the Choroid," Ref. Soc. Study Dis. Child., London, 1901, i, 169–181.
4. Marple, W. B.: "Tubercle of the Choroid in Tuberculous Meningitis," Ophthalmoscope, London, 1912, x, 559–563.
5. Heinzel, L.: "Ueber den diagnostischen Werth des Augenspiegelbefundes bei intracraniellen Erkrankungen der Kinder," Jahrb. f. Kinderh., Leipzig, 1875, N. F., viii, 331–359.
6. Manz, W.: "Tuberculose der Chorioidea," Arch. f. Ophth., Berlin, 1858, iv, part 2, 120–126.
7. Manz, W.: "Tuberculose der Chorioidea," Arch. f. Ophth., Berlin, 1863, ix, part 3, 133–144.
8. Busch, F.: "Zwei Fälle von Geschwulstbildung im Augenhintergrunde," Virchow's Arch., Berlin, 1866, xxxvi, 448–454.
9. Cohnheim, J.: "Über Tuberculose der Chorioidea," Virchows Arch., Berlin, 1867, xxxix, 49–69.
10. Litten, M.: "Ueber Acute Miliartuberculose," In: Samml. klin. Vortr., Leipzig, 1877, No. 119 (Inn. Med., No. 43, 1061–1098).

DISCUSSION.

Dr. S. D. Risley, Philadelphia, said he did not know of anything in his medical experience which had caused him more surprise than the frequency with which he had found tuberculosis as an etiologic factor in uveal disease, after his attention had once been directed to the subject. He was convinced that for many years he had been overlooking the actual etiology of many cases of choroiditis and iritis, ascribing them to other and various causes, which he now believes had in all probability been tubercular in character. Cases of progressing disseminated choroiditis he had repeatedly found responding positively to tubercular tests, and their progress arrested by gradually ascending doses of old tuberculin by hypodermic injection after other methods of treat-

ment had entirely failed. As illustrating this claim, he had seen a sudden onset of edema occupying a large area in the nasal ophthalmoscopic field in a patient who had long been under treatment for weak eyes, with irritable eye-grounds and a low astigmatism with hyperphoria. In the center of the edematous area could be seen a dense, gray-white spot. The von Pirquet test proved very positive, increasing the irritability of the eye and causing a slight rise in temperature. Ascending doses of old tuberculin were resorted to, with the result that the edema rapidly disappeared and the vision rose from one-fifth to normal, but as a final result there was a large patch left in the choroid similar to those with which we were familiar in the frequent cases of disseminated choroiditis. He quite agreed with Dr. Randolph as to the necessity for more frequent study of eye-ground lesions, especially in the presence of kidney affections. In his experience it had been rare not to find changes in the fundus in cases of advanced nephritis. He was not ready, however, to believe that the eye-ground lesions were necessarily due to the kidney disease, but that both nephritis and the retinitis were due to a common toxemia, which was to say that the retinitis was but another local expression of a general or systemic disorder which involved also the kidney; as, for example, in glycosuria or cardiovascular disease, related as they are to faulty metabolism.

DR. R. L. RANDOLPH, Baltimore, Md.: I would like to say that these miliary tuberculoses of the choroid differ entirely from anything we see in so-called disseminate choroiditis. The tubercle, for instance, shown in the drawing is distinctly round and elevated above the plane of the retina, and there is no suggestion of any pigment about it, a thing which always occurs in choroiditis disseminata.

I have great faith in the tuberculin and Wassermann tests, but we so frequently find contradictions in their exhibition that I am not disposed to place unbounded confidence in them. I am not so sure that cases should be regarded as cases of tuberculosis simply because they get well promptly under the treatment usually adopted for tuberculosis.

REPORT OF CASES OF QUININ AMBLYOPIA, WITH REMARKS.

JOHN E. WEEKS, M.D.,
New York.

Although quinin amblyopia is well known to all ophthalmologists, the cases here reported possess some features that, in the opinion of the writer, justify their being presented here.

Case I.—Mrs. H. K., aged fifty-one years. On January 19, 1916, the patient was operated on for fistula in ano and hemorrhoids while under a general anesthetic. It was thought advisable to give 20 grains of quinin per rectum after the operation to control pain (as a nerve block) and a prescription was sent to a druggist. Instead of writing the prescription in troy weight, it was written in the metric system, as follows:

℞. Quininæ muriate..... 20.00
Aquæ.....120.00

—an amount equivalent to about 315 grains of quinin. The 120 c.c. was injected well up into the rectum by means of a syringe and a soft-rubber catheter. The solution was quite warm when the process of injecting it began, but cooled somewhat as the injection proceeded. As the preparation cooled, crystallization occurred because of oversaturation, partly clogging the catheter. Some of the preparation escaped from the rectum. Probably about one-half of the quinin was retained. The quinin was injected about 6.30 P. M. Some morphin had been given hypodermatically before the operation. Incessant vomiting occurred from 9 P. M. to 1 A. M. The stomach was washed out with a solution of sodium bicarbonate, and $\frac{1}{30}$ grain of strychnin sulphate was given hypodermatically some time before 6

A. M. on January 20th. At 9 A. M. the patient opened her eyes and inquired if there was a light in the room. Her physician became aware that vision was abolished at 10 A. M. At that time the pupils were dilated three-fourths maximum. Right iris was not responsive to light; left iris responded, but almost imperceptibly. Vision nil. Patient complained of tinnitus and deafness. Blood-pressure (systolic), 120. Heart action normal. Patient was seen by the writer at 2.15 P. M., twenty hours after the quinin had been given, who, after hearing the history, made a tentative diagnosis of quinin amblyopia, which was confirmed on examination of the eyes.

Status Præsens.—Pupils dilated. Reaction of irides to light and vision as at 10 A. M. Optic discs very pale; capillaries empty. Veins and arteries about two-thirds normal size. Volume of blood apparently not more than one-half the normal. Caliber of vessels uniform. Vessel walls transparent. Retina edematous and pale throughout, except at the macula, the edema being almost as dense at the periphery as in the other parts of the retina. There is no very well-defined "cherry red" spot, but there is very little edema at the fovea centralis, this area having a brownish-red appearance. Ocular motility normal. Patient prostrated by the nausea and vomiting and the effects of the anesthesia.

The husband was very anxious to know the probable outcome regarding vision. He was informed that vision would return, but to what degree could not then be determined.

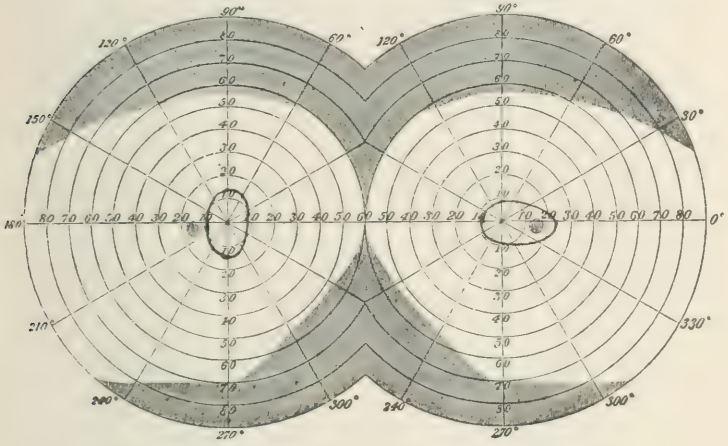
Treatment, which was instituted at once, consisted of a high, copious, saline enema (two quarts), repeated in two hours and again four hours later. This was advised in order to wash out any quinin that might have remained in the bowel. Glonoin, $\frac{1}{100}$ grain, every three hours, for the purpose of relieving vascular spasm. Strychnin, $\frac{1}{40}$ grain, three times daily (the glonoin and strychnin hypodermatically) as a sustaining measure.

At five o'clock of the same day the retinal blood-vessels were not quite so small, and the edema of the retina at the periphery was not so marked.

January 21st, 8 A. M.: No perception of light. Edema of

retina less marked. 3 P. M.: Pupils react very slightly to stimulus of light. Patient can distinguish bright light, as the flash from an electric bulb. 8.15 P. M.: Consultation with Dr. George E. de Schweinitz, who suggested giving digitalis in addition to the other remedies mentioned, with the idea of increasing the force of the blood-current to some extent. Digipuratum was given in one-grain dose three times daily.

January 22d, two o'clock P. M.: Blood-vessels of retina appear to be a little larger. Periphery of retina clearing. Irides react more readily to stimulus of light. Can see hand movements.



Case I.—Fig. 1. H. K. January 27, 1916.

January 23d: Sees to count fingers at four inches. Slight capillary flushing of inner upper quadrants of discs. Retinal blood-vessels about two-thirds normal size—regular. Glonoin reduced to $\frac{1}{200}$ thrice daily. Digipuratum and strychnin continued.

January 24th: Digipuratum not well borne, discontinued. Glonoin discontinued.

January 25th: Urine examined qualitatively. Trace of albumin. No sugar, no casts. No trace of quinin.

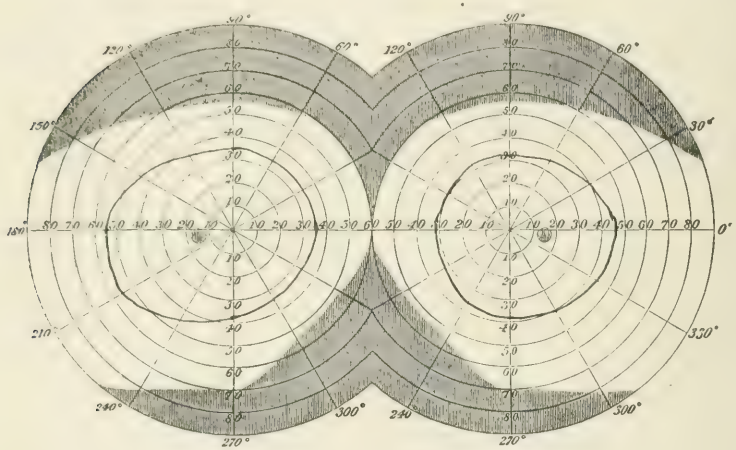
January 27th: Fields of vision taken. (See Fig. 1.)

Sees colors correctly when in large surface. The margins of the retinal vessels show as narrow white lines.

January 30th: Vision, R. E., 20 30 −, L. E., 20/30, central.

January 31st: Blood-vessels not so large as on January 24th. The strychnin and glonoin were renewed. The lumen of the arteries has become irregular; the walls of the arteries in particular appear to be irregularly thickened, this being apparent by a difference in the width of the bordering white lines.

February 2d: As the blood column in the retinal arteries



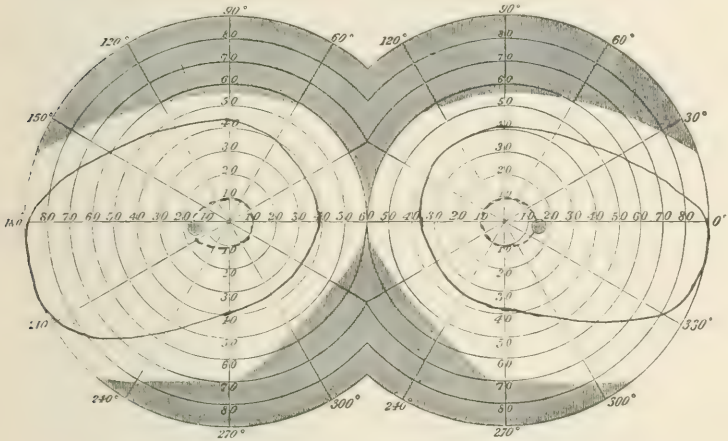
Case I.—Fig. 2. H. K. February 26, 1916.

appeared to have diminished in size, it was thought desirable to find some means to increase the blood-flow, and in this quest Dr. Evan Evans, internist, was called in consultation. Dr. Evans expressed the opinion that it would be impossible to increase the blood-pressure for any length of time, if at all, by any means, with a normally functionating heart, but suggested caffein in addition to the glonoin and strychnin (caffein, 2 grains; glonoin, $\frac{1}{100}$ grain, given every four hours, the strychnin as before). Some improvement in the size of the retinal blood-vessels followed and the irregularity in the lumen of the arteries slowly disappeared.

February 4th: Began to take the iodid of strontium for its effect on exudation. Both discs show an increase in the capillary circulation. Vision of each eye central 15/20.

February 7th: Dr. O. Schirmer saw the patient in consultation and suggested massage by means of an electric vibrator to be applied once or twice daily. It was thought that the use of the vibrator might endanger the integrity of the eyes by inducing hemorrhage or detachment of the retina. As a substitute, gentle digital massage was instituted.

February 13th: Condition of discs and retinal vessels



Case I.—Fig. 3. H. K. March 26, 1916.

better. The irregularity in the thickness of the walls of the retinal arteries has disappeared, the calibers now being uniform.

February 26th: Fields of vision as in Fig. 2.

February 29th: Dr. de Schweinitz, who saw the patient at his office, reported the light perception, as tested by Snellen's photometric letters, to be 75 per cent.

March 26th: The optic discs present about two-fifths normal capillarity. Arteries one-half normal size. Veins slightly narrower than normal. Fields of vision as in Fig. 3. Parinaud's photometric bars indicate a light perception

of 80 per cent. Vision, 20 20 – in each eye. Patient reads ordinary type readily.

April 10th: The patient's general condition is somewhat below normal. While her weight is normal, the patient becomes fatigued easily, suffers from insomnia, and is in a more or less depressed condition mentally.

Throughout the entire progress of the case, since light perception began, the patient, while able to see objects, has complained that she "cannot see the light," apparently meaning that the light, particularly daylight, does not have the ordinary intensity. With virtually normal vision the patient continues to inquire, "When shall I see the light?" or "When shall I see daylight again? Everything is so dark." There does not appear to be any great amount of torpidity of the retina, as objects are almost normally visible when the light is modified, but if the light is considerably reduced, the reduction in light perception causes her to lose form before it is lost to the normal eye. I am of the opinion that the phenomena that she so constantly complains of are due to the reduction in the light sense.

April 26th: Patient died from injuries received as the result of a fall.

CASE II.—Miss Rose L., aged sixteen years, entered the hospital at the Rockefeller Institute December 13, 1915, having developed pneumonia two days previously.

[The selective action of certain of the salts of quinin against the pneumococcus is now well recognized by clinicians. One of the most active is ethylhydrocuprin (optochin), produced by Zimmer & Co., of Frankfort, Germany. Studies have been conducted at the Rockefeller Institute recently to ascertain the therapeutic value of ethylhydrocuprin in the treatment of pneumonia. To January 31, 1916, eleven selected cases had been treated. Ocular symptoms developed in but one.]

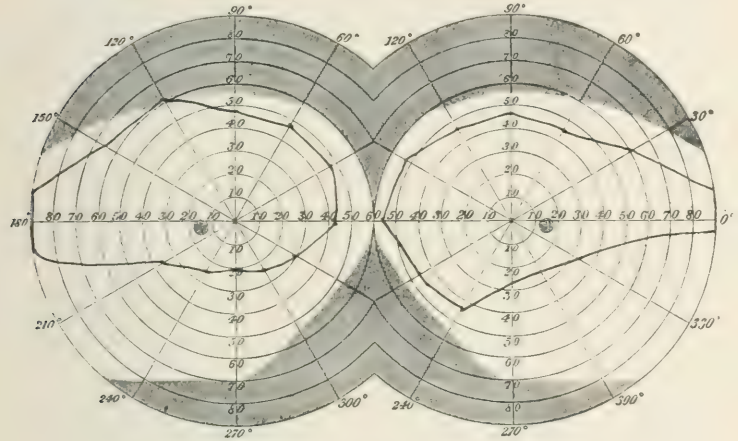
December 14th: Ethylhydrocuprin (optochin) was given by mouth in 0.5 gram dose, repeated every eight hours until two grams were taken. The last dose was at 5 P. M., December 15th. At 9 P. M. the patient complained that she could not see.

December 21st: Patient began to distinguish light.

Vision slowly improved from this time until, at the end of six weeks, it equaled 20/30 − in each eye.

January 27, 1916: By the courtesy of the physician in charge I was privileged to see the patient.

Status Præsens.—Irides react readily to the stimulus of light. Vision, 20 30− in both eyes. Fields for form: Right eye, above, 20°; below, 15°; nasally, 15°; temporally, 35°. Left eye, above, 25°; below, 25°; nasally, 20°; temporally, 35°. Optic discs paler than normal, but the pinkish flush (capillarity) was fairly well marked; veins very nearly of normal size; arteries one-half the normal size; walls of



Case II.—Fig. 4. R. L. February 13, 1916.

blood-vessels thickened, showing as white lines bordering the blood column. The white lines, particularly along the arteries, were not of uniform width, causing the blood column to appear narrower at some places than at others.

February 13, 1916 (about two months after the ethylhydrocuprin was taken). Fields of vision for form as shown in Fig. 4.

The patient's general condition is excellent.

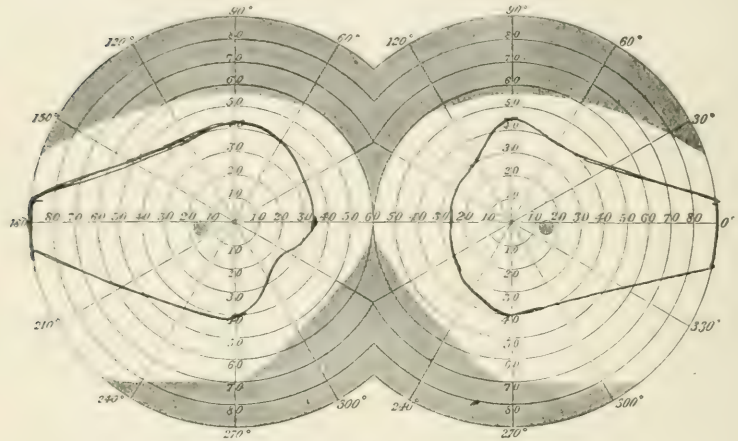
May 6, 1916. Vision of each eye, 20/30−.

Fields of vision, as in Fig. 5, slightly reduced, particularly to the nasal side from the field as of February 13th. L ght

perception approximately 80. Color vision very indefinite. Many of the retinal arteries are greatly reduced in caliber, their walls irregularly thickened, the optic discs considerably paler than usual, but not extremely pale. The condition of the retinæ does not seem to be as good as on January 27th.

CASE III.—The following is a supplementary report of a case first reported by the late Dr. Herman Knapp in the *Archives of Ophthalmology* (vol. x, 1881, p. 220):

"Patient, Jennie L., age six years. Chill on the afternoon



Case II.—Fig. 5. R. L. May 6, 1916.

of April 3, 1877. April 4th, A. M., temp. 106¼° F., pulse 150. Ordered quinin, 10 grains, by rectum. At 9 P. M. ordered quinin 2 grains every hour by mouth. April 5th: Quinin by mouth having been rejected, has been taking it by suppository, 10 grains every four hours, nearly.

"April 6th: Took 10 grains of quinin by enema last night. Ordered 10 grains of quinin to be given by rectum at noon. 4 P. M. restless. Pupils dilated, irides immovable. Vision abolished. More or less quinin given to April 10th.

"April 11th: Patient saw sparks of fire from the open grate."

Dr. Knapp saw the patient for the first time February 16, 1878, almost a year after the quinin had been taken. He reports:

"When she presented herself to me, she was in good general health. She read easily Sn. xx at 20′, and 0.25 at 8″, with each eye, but showed a moderate concentric limitation of the visual field in each eye. Her color-perception was normal. The ophthalmoscope showed a striking picture: both optic discs white, the retinal vessels scant and very small, especially the arteries. The pupils were of normal size and mobility, the eyeballs of normal tension, showing nothing unusual in external appearance and mobility.

"On May 11, 1879, she presented herself to me again. Four months previously she had had malaria, and taken quinin again; no more, however, than 10 grains in one day. The parents had noticed that from that time her sight was impaired. I found S. 20 100 in the right, and 26 100 in the left. She read 0.50 with difficulty. No improvement with glasses. She was green-blind. Her field of vision was very much contracted; in the right eye almost to the point of fixation, in the left having a diameter of 15°, of which 5° were on the nasal, 10° on the temporal side, from the point of fixation. The optic discs were white; the blood-vessels, especially the arteries, few and very small. Patient had frequently suffered from headache.

"In May, 1881, while preparing this paper, I examined her again. Her sight was 20 30 in each eye. Emmetropia. She experienced no inconvenience from her eyes in any way. Her perception of colors, tested with different methods—selection of samples, pseudo-isochromatic plates, contrast-shadows—proved perfect. Her light-sense was normal. She never complained of seeing less in the dusk than other people, and was able to read the same type as persons with good eyes, when I gradually obscured the examination-room. Her field of vision, however, was still contracted, and, measured perimetrically, showed almost symmetric figures in both eyes.

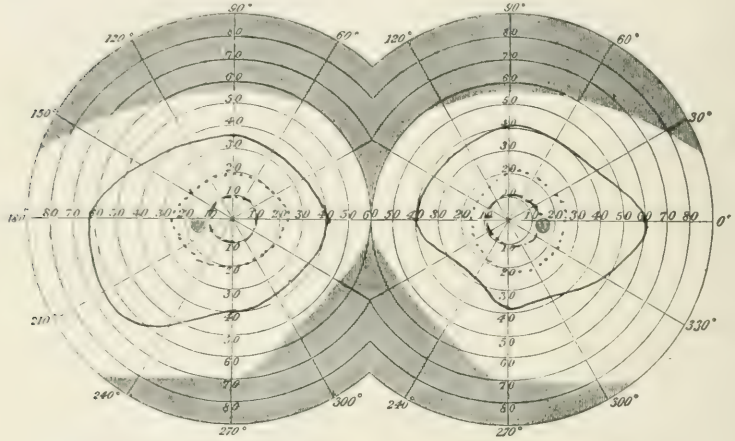
"The optic discs were white, with a faint pinkish tinge. Both arteries and veins were few and small, but none were white. The choroidal vessels, clearly visible, were normal in color, size, and distribution. The appearance of the

fundus oculi, in general, was normal, with the exception of the changes of the optic disc and retinal vessels, as described above."

The patient was seen by the writer on February 26, 1916, and on May 19, 1916.

A vertical nystagmus is present in both eyes. Vision with correction in right eye was 20 30; left eye, 20 40 −. The fields of vision as in Fig. 6.

The optic discs are almost chalky white. The blood-vessels were few in number and much reduced in size. The arteries in particular are very narrow. The walls of the vessels do not show marked thickening. The fundus



Case III.—Fig. 6. J. L. May 19, 1916.

in general presents the appearance that accompanies a reduced blood supply, not as vivid a reddish brown as is found in the normal fundus. It is evident that the nystagmus has developed since the patient was seen by Dr. Knapp thirty-eight years ago, and that the vision has deteriorated slightly. The fields of vision are reduced but little. The only symptom referable to a diminished light perception is experienced when passing from a well-lighted to a faintly lighted room, when, although the irides are active, some time is necessary for adaptation. On the whole, it is remarkable that so little change has taken place after the long lapse of time.

In regard to the cherry-red spot. In the case of Mrs. H. K. the area of the fovea centralis, while it presented a reddish color on account of the absence of edema at this point, did not show the vivid cherry-red color so generally found in obstruction of the central artery of the retina, the reason for this being, if my interpretation is correct, an ischemia of the choroid as well as of the retina, due to the effect of the quinin on the vessels of the choroid, the absence of a free flow of arterial blood in the choriocapillaris causing the appearance at the fovea to lose much of its vividness and to have a reddish brown color.

The tension in the three cases noted was normal.

In one of Roosa's (Knapp's *Archives*, vol. ix, p. 41) cases which he saw two years after the amblyopia occurred the patient complained that she saw as though a veil was over her eyes—"cannot tell whether her linen is clean when it comes from the wash." Vision at this time = 20/40 O. U. In our first case the complaint was not of a sensation as though she was looking through a veil, but as though the light was not of sufficient intensity. Objects were not sufficiently well illuminated. This was on account of a 15 to 20 per cent. loss of light perception.

Treatment: Inhalations of nitrite of amyl, digitalis, strychnin, galvanism, hydrobromic acid, bromid of sodium, and the iodid of potash are remedies mentioned. Von Graefe depleted the patient. It would seem to the writer that the indications for treatment are first to relieve and to prevent vascular contraction. Remedies that will do this should be employed. Vigorous supporting measures in the way of diet and pure air should be instituted. For the first week or ten days after the amblyopia is induced it is probable that the recumbent position with the head low may be of value as favoring a greater flow of blood through retinal and choroidal vessels.

DISCUSSION.

DR. H. D. BRUNS, New Orleans, La.: While no one could add anything to the thorough study of the cases which Dr. Weeks has presented, I think it may be of interest to communicate the fact that I have seen two cases after very long intervals, having observed them at the time of the blindness. I have seen many cases of quinin poisoning, owing to the fact that I live in a region where malaria is common. There seems to be a great individual difference in susceptibility to the drug. We constantly see patients who have taken, especially since the researches at the Isthmus, large doses for three days in succession without having suffered any ill effects. On the other hand, we see patients afflicted with blindness from comparatively small doses. Of the two cases that I saw after a long interval, one was a child six years of age who presented the ordinary symptoms, easily recognizable. I saw this patient afterward when she was a grown woman and had children of her own, and the singular thing was the absolute incongruity of the sharpness of vision with the ophthalmoscopic appearance.

The other case was that of a man who was treated with quinin in about 1867, I think, for yellow fever. At that time they were experimenting with quinin as a remedy for yellow fever, and he had been made blind. I saw him perhaps twenty-five years after that time. In both of these cases no ophthalmoscopist would have failed to make a diagnosis of advanced optic atrophy. The disc was white and the vessels were very small, and yet the vision, with correction, in both cases was certainly better than 20 30 in each eye. In both cases there was decided contraction of the fields of vision. I would say that the field of vision—of course I do not remember the details with absolute accuracy—was about two-thirds of the normal. Reports of both of these cases were published, I think, in the *New Orleans Medical and Surgical Journal* some years ago.

DR. E. C. ELLETT, Memphis, Tenn.: I live in the same malarial district as Dr. Bruns, and they give quinin down there with a shovel. I have seen some of these cases, and from my experience I get the same impression of idiosyncrasy and also as to the preservation of the central vision.

I would like to present the report of seven cases, the ages of which varied from seventeen months to fifty-nine years, the incidence of the quinin amblyopia varying from seventeen months to twenty-eight years. The longest time after the onset of the trouble that I have observed a case was thirty-four years after the amblyopia came on. The doses in these cases varied from eight grains to one ounce, the eight grains being given in one dose, and it happens that in all of the others the quinin was given at short intervals—within a day or two. The blindness came on in every case, where I could ascertain, suddenly, and it has been likened to the turning off of the light. It persisted from one week to three months, and all of the cases ultimately showed the ophthalmoscopic appearance of optic atrophy with great contraction of the vessels and narrowed visual fields, and some showed paracentral or ring scotoma; none central scotoma. The ultimate vision in these cases was from 1/200 to 20/20. My impression from these cases is that they do as well without as with any treatment we know of.

CASE I.—F. F., male, aged thirty years. Seen in 1897. Three years before that he had taken 120 grs. of quinin in twelve doses in twelve hours. Became deaf in four days, lost sight rather suddenly. Was blind three months. Vision 5/200 and 1/200. Ophthalmoscopic appearances of optic atrophy. Vision improved by amyl nitrite. Fields: R. E., large scotoma down and out; slight contraction; L. E., contracted field and central scotoma.

CASE II.—G. R., aged nine years, seen May, 1912. When two and one-half years old was given 300 or 400 grs. of quinin hypodermically within a week. Was blind six weeks. Vision with glasses 20/40 p. and J. 3. Fields contracted to 10°. Optic nerves atrophic; arteries are white cords; veins few and small, with fine superficial pigment dots near the disc, somewhat like that of retinitis pigmentosa.

CASE III.—Mrs. W., aged fifty-nine years. Seen October, 1915. Twenty-one years before had taken a total of 120 grs. of quinin. Vision 20/25 and 20/20, with glasses. Fields contracted; R. E., up and down 15, in and out 30; L. E., not quite so much. Nerves white, arteries carry blood, but have wide, white bands following them.

CASE IV.—A., aged seventeen months, seen in October, 1910; 2 grs. of amorphous quinin by mouth every three hours until 24 grs. were taken, when she suddenly went blind. The quinin was continued several days and she was given a hypodermic of quinin and urea. Was seen one week after onset of blindness. Eye-grounds normal. Myopia, 8 and 12 D. Four days later the nerves were pale and vessels small. Three weeks later, or four weeks after onset of trouble, the refraction was Em., and apparently central vision was present. Six weeks after the sight was lost it was good again. In 1914 the nerves were atrophic, vessels small, refraction Em.

CASE V.—R. W., aged twenty-four years. November, 1913. Was given 5 grs. quinin every three hours for three days by father, then 15 grs. hypodermically by physician. About August 24th became blind and remained so one week. The patient says he received two hypodermics in one day, and loss of vision followed at once. There was moderate atrophy and narrow, white-bordered vessels. Fields irregularly contracted and paracentral scotoma. Vision 4/200 each eye. Two months later vision 18/65 each eye.

CASE VI.—W. N., aged forty-nine years. Seen 1912. When fifteen years old took one ounce quinin in three or four days. Was blind ten days, when vision returned, and was serviceable until he developed cataract. The discs were papery white and the vessels hardly visible. Fields showed ring scotoma in each eye.

CASE VII.—L. P., aged six years. Seen September, 1915. On August 30th he had violent convulsion and rectal temperature of $105\frac{1}{2}^{\circ}$. The blood showed severe tertian malaria and he was given 8 or 9 grs. of quinin hypodermically. He regained consciousness in twenty-four hours, but was blind. No more quinin was given and the fever subsided in six days. At that time he saw light, and vision improved until September 11th, when he saw 15/20 in each eye. Nerves slightly pale, and slight contraction of vessels. Fields could not be taken.

DR. CARL FISHER, Rochester, Minn.: In view of the marked decrease in the size of the retinal arteries it has

seemed to me that amyl nitrite might be valuable in the treatment, and I wondered if Dr. Weeks had had experience with it or does he see any objection to such treatment?

DR. G. E. DE SCHWEINITZ, Philadelphia: The relation of idiosyncrasy to quinin blindness is well recognized, as indeed it is in several forms of toxic blindness, notably that produced by methyl alcohol. The smallest dose of quinin that I have ever seen produce amblyopia, very temporary in character, was 12 grains administered in twenty-four hours. Dr. Weeks refers to the changes which have been observed in the retinal vessels in patients with quinin amblyopia. These ophthalmoscopic evidences of retinal vascular disease in the sense of obliteration of the arteries and apparent conversion of their walls into white cords in long-standing quinin blindness, either in human beings or in animals, has been confirmed by a number of observations, beginning with those of Horner, and this observation I have myself made in my experimental work, as well as in clinical researches. At least one case is on record: namely, Parker's, in which thrombi were believed to have been seen in the inferior retinal vein as early as four days after the blindness. But it has not been proved that such vascular alterations constitute the underlying basis of the amblyopia, and even in long-standing quinin blindness in animals it is doubtful whether the presence of any true endovasculitis has been demonstrated. Certainly the thrombus formation which I described many years ago represented in all probability an incorrect description, and depended upon a faulty interpretation of the phenomena observed. In this connection, however, it is interesting to refer to a comparatively recent Continental writer, who has expressed the opinion that these very vascular changes which are clinically evident have an important relationship to the development of this type of toxic blindness, and in this paper he refers to the fact, quoting some of my own earlier observations, that "the older writers" were correct in their observation and belief that vessel alterations were of etiologic significance.

DR. J. E. WEEKS, New York: In regard to the cherry spot, I do not think that it exists, as we see it in occlusion of

the central artery of the retina, for the reasons given in the text of the paper.

In the treatment of the case reported, glonoin was employed because of its persistent effect on the size of the blood-vessels. Amyl nitrite may be employed with advantage; however, its effect is more transient than that of glonoin.

RETROBULBAR NEURITIS WITH CENTRAL SCOTOMA FROM TOXIC ACTION OF THYROIDIN.

MYLES STANDISH, M.D.,
Boston, Mass.

Thyroidin and thyroid extracts have attained so large a place in our armamentarium that if they are capable of producing optic neuritis involving the macular bundle of fibers it seems desirable that this fact should be well established.

The number of cases reported to date, considering the increasing use of the drug in the last few years, has been very small. It is possible that, with other conditions present, neuritis has been overlooked.

I have been able to find but eight cases of retrobulbar neuritis with central scotoma which have been attributed to the use of thyroid extracts.

Dr. Henri Coppez* reports five cases. There were four women and a man between thirty and forty years of age. They developed the toxemia after using the drug in large doses for some months. They had all lost a large amount in weight, but the only other symptoms were general nervous disturbances. There was in each case a loss of vision in both eyes to 1/10 or less, but not an equal amount in each eye.

Dr. Coppez reports one case in full: Man, thirty-six years of age, had been taking thyroid tablets in large doses and had lost in eight months 26 kilograms in weight. The latter

* Archiv. d'Ophtal., 1900.

part of this time he had taken nine tablets daily. The general conditions were nervousness, sleeplessness, and muscular weakness. Ophthalmoscopically he had hyperemic nerves without inflammation, there was venous stasis in the retinal vessels, and slight pain from pressure on the globe. V. R. E. =1/12; L. E. =1/6. Absolute central scotoma of about 10° in the right eye and a relative scotoma in the left eye. The periphery of the field was intact. He was forbidden thyroid, alcohol, and tobacco. After three months vision in the right eye was ¼ and in the left ½, and he had in the right eye a slight central scotoma for red and green. The periphery of the field for white and color remained normal. After three months vision in the right eye was 1/3 and in the left eye 2/3. The body weight had increased. The retinal veins were still slightly turgescent. It is unfortunate that this was the case of the man, and that alcohol and tobacco were forbidden coincidentally with thyroidin. Coppez says that the discs were somewhat more altered in appearance than in alcohol and tobacco neuritis. The veins were often swollen and the arteries diminished in size.

Carl R. Hennicke* reports a case of a man fifty-three years of age. First seen in 1907. He had a central vision of 6/9 in each eye, with a myopia of 0.50 diopter. The ophthalmoscope showed the left eye with normal fundus. The right disc was somewhat reddened. The outline of the nerve was not lost. The arteries were very small, veins of normal size. It was a case of myxedema and had been under treatment five years. In June, 1910, vision in the right eye was 6/30. Ophthalmoscopically as in 1907. The patient had taken thyroidin since 1903. He did not return till September 21st. Vision in the right eye was then movements of the hand, and in the left eye was 6/24. The right nerve was white, the arteries scarcely visible. The left disc was swollen and reddened and the outline blurred. The neighborhood of the nerve not clear, the arteries small, and the veins swollen. Iodid of potash was prescribed and thyroidin was omitted, and on November 21st vision in the right eye was 6/30 and in the left 6/18. Hennicke thought the ophthalmic changes were not due to myxedema, but to thyroidin. On January 27, 1911, vision in the right eye was 6/30 and in the left 6/12.

* Klin. Monatsb. f. Augenh., 1911.

Dr. Emanuel Snydacher, in a clinical note,* reports a case of a woman forty-two years of age who came in complaining that in bright light she had difficulty in seeing, all objects appearing more or less hazy; but in a subdued light, or even in comparative darkness, she had no trouble. An ophthalmoscopic examination showed an apparently normal fundus, with the possible exception of slight blurring of the edges of the disc. Vision was 6/9 in each eye.

Perimetric examination disclosed an oval central scotoma for red and green in both eyes. Investigation of her mode of life revealed no apparent cause for this condition. She denied being addicted to either tobacco or alcohol. Repeated examinations disclosed no sugar in the urine, this being a frequent cause of a chronic toxic amblyopia, such as she was suffering from.

Quite accidentally, during a subsequent examination, she volunteered the information that, for a number of months, she had been taking an antifat remedy recommended by a friend. Investigation revealed the fact that this patient had been taking thyroid extract for almost three months to reduce her weight. The thyroid was at once stopped, the patient was given strychnin hypodermically, and in a few weeks the scotoma disappeared.

Aalbertsberg† reports a case of optic neuritis following the use of thyroid gland in a patient with myxedema who had been treated with thyroid gland. The effect on the disease was favorable, but the patient began to complain of poor vision in the left eye. Examination revealed optic neuritis. Later vision failed in the right eye and a low degree of neuritis was found here. The use of thyroid was stopped immediately and the condition of the eye improved. In the left eye, however, an atrophy of the nerve came on.

On May 7, 1910, a woman, aged sixty-nine years, came to see me who had discovered a short time before that vision in the left eye was not so good as in the right. She had a small central scotoma in the left eye and a central color scotoma in the right eye for green. Her vision was 6/30 in the right

* Jour. Amer. Med. Assoc., October 28, 1911, p. 1473.

† Weekbl. v. het. Ned. Tijdschr. v. Geneesk., ii, No. 22.

eye and 6/25 in the left eye. About ten days before she had first been unable to read newspapers.

With the ophthalmoscope there was found some slight swelling of the optic disc in the right eye, some narrowing of the arteries, but not marked turgescence of the retinal veins. There were several small retinal hemorrhages in each eye. It was originally a case of myxedema, and I was told that formerly she had the enlarged features and club-fingers which are markedly characteristic of the disease. When she came to me these characteristics had disappeared. She was anemic, emaciated, weak, nervous, and looked like a very ill woman. She had been treated with thyroidin, and after several years' treatment the recovery from the myxedema was considered complete. However, she continued the drug on her own responsibility after ceasing to visit the doctor. For fourteen years she had taken a 5-grain thyroidin tablet three times a day, and for five or six years had taken one tablet each day.

Examination failed to find any disorder of the kidneys, and the physician in charge was inclined to attribute the retinal hemorrhages to her marked anemia. Thyroidin treatment was discontinued and vision improved. I did not see her again, but was told the improvement was sufficient to enable her to thread a coarse needle, but there was a recurrence of the myxedemic symptoms. She died suddenly on July 19th.

On September 11, 1911, a man, aged forty-one years, was referred to me who had noticed on the previous January a blur before his right eye, and at the same time thought the vision in his left eye was not as good as formerly, and vision had continued to decrease. It was then 1/10 in the right eye and 8/10 in the left eye.

Six years before he had had one-half of the thyroid gland removed on account of a fibrous tumor, and four years afterward was supposed to have had the other half removed for the same cause. After the last operation he had a pulse of 120; after the first operation there had been no increase of pulse. The outer limits of the field in the right eye were normal. He had a central color scotoma in the right eye for all colors except dark blue. He was slightly more excitable than formerly, and had some uncertainty in walking. There

was no evidence of kidney disease or diabetes and no history of syphilis. No abnormal pupil symptoms. He had taken since his last operation ½ grain tablet of thyroidin before each meal.

Ophthalmoscopically the nerve was normal in color in each eye and vessels not markedly changed. He was advised to omit thyroidin, and his vision returned in a few months to normal in each eye. There has been a return of the tumor of the thyroid, but his general condition is excellent.

On December 23, 1913, a woman, who had been under my care eleven years for other ocular conditions, came to my office complaining of sudden loss of vision. She was thirty-three years of age, stout, and on advice of a friend had attempted to reduce her weight by taking a proprietery medicine. The composite name implied that it contained thyroidin. The medicine was put up in the form of a bolus which weighed 10 grains. The only analysis of the medicine that I was able to find would seem to show that the bulk of the mass was probably desiccated thyroid. The label on the parcel said that the medicine was "designed for the treatment and reduction of excessive and abnormal fatty accumulations on the human body, and for the reduction of goiter and glandular enlargements."

She had begun taking this five days before; had taken three doses on the eighteenth, three on the nineteenth, and six on the twentieth. The following morning her vision was so reduced that she could not read and had remained so since.

Ophthalmoscopically both nerves were pale and the arteries small. There was a central scotoma in the right eye for all colors, except a somewhat uncertain perception for blue. In the left eye the central scotoma was complete for all colors. There was no headache nor nausea. Vision of each eye was 20/200. The pulse was 120. The nostrum was omitted, and on December 26th the pulse was 100, full, and easily compressible. On January 26th, which was the last time I saw her, vision in the right eye was 20/40 and in the left eye 20/70, and the pulse 90.

Ophthalmoscopically both discs were somewhat whiter than normal, especially the left.

Birch-Hirschfeld and Inouye* state that experimental investigation on dogs shows the anatomic changes to be a degeneration of the ganglion-cells of the retina with secondary degeneration of the optic nerve-fibers.

It seems probable that in these three cases the retrobulbar neuritis was due to the thyroidin, as in each case the discontinuance of the drug was followed by a recovery of vision.

The most startling thing in this short series of cases is the wide difference in the toleration of the drug. One patient had taken the thyroidin in large doses for many years, and one patient had taken an unknown amount for only three days. That the susceptibility to toxemias varies greatly in different individuals, and indeed in the same individual at different times, is well known. In the case of tobacco a long series of years' use without harm may precede the toxic neuritis. The rapid production of the toxemia in the last case is occasionally paralleled in other drugs.

I have hesitated about reporting the last case owing to the fact that one does not expect pathologic changes of this sort to be due to an acute poisoning from drugs, but where so little is known of this toxic action on the optic nerve, it seems advisable that all cases in which the condition is suspected should be placed upon record. My principal object in reporting these cases is to place them on record, and to sound a word of warning that thyroidin should not be used indiscriminately for reduction of weight, and that the selling of proprietary medicines containing it to the public for this purpose is very likely a dangerous practice.

* Graefe's Arch., lxi, p. 3.

OPTIC NEURITIS FROM SYPHILITIC LEPTOMENINGITIS.

WILLIAM H. WILDER, M.D.,
Chicago, Ill.

Syphilitic infection of the membranes of the brain most frequently occurs in the arachnoid and pia mater. Syphilis may affect the dura and cause pachymeningitis, but it is the condition brought about by leptomeningitis that affects so seriously the nerves of the eye, both optic and motor. While this disease has generally been regarded as a late or tertiary manifestation of syphilis, numerous observations have shown that it may appear in the so-called secondary stage of the infection, and even a few months after the initial lesion. Many writers on the subject designate the disease as gummatous meningitis, applying this term to all forms of syphilitic meningitis, but this designation tends to give an incorrect impression of the pathologic findings on autopsy. In addition to focal lesions there may be localized exudates, areas of hyperplastic inflammation, and vascular changes.

According to Ziegler,* there first develops in the pia and subarachnoid tissue a circumscribed inflammation, which gradually leads to the formation of a gray or grayish-red granulation mass rich in cells and containing new vessels. This mass may fill in the space between the sulci; some of it may become organized, while other parts, for instance, the center, may undergo caseation.

The brain substance adjoining such a gummatous formation may also become involved in the process. Such gummatous masses may vary in size from those invisible to the

* Ziegler: Pathologische Anatomie, Bd. ii, p. 367.

naked eye up to nodules as large as a walnut. The smallest may be seen as points of thickening of the arterial vessel-walls. Smaller masses may be absorbed; larger ones may become partly organized or undergo caseation.

Should the inflammation affect the membranes of the convexity, these gummatous masses fill in the sulci and conform to these spaces. Most writers state that the basilar meninges are more frequently affected than those of the convexity.

LeCount,* however, from an examination of 55 brains with syphilitic meningeal lesions, thinks it probable that the membranes of the vertex are as frequently affected as those of the base. From his observations he describes three main types of changes in the pia—opacity or turbidity, patches of fibrous thickening, and discrete focal lesions. He found turbidity of the pia in some degree and extent in nearly all cases examined by him. He found this turbidity extending over almost the entire brain, usually with the exception of the occipital lobes, shrouding it as with a veil or sheet. Sometimes it appeared only in patches, and often the base was entirely free, but, when found there, it was well marked over the pons, the interpeduncular space, and the region of the oculomotorius. When the opacity was prominent the brain, if fresh, had a bluish white, dull hue, and appeared as if milk had been poured over it.

In the second type of alteration, together with turbidity of the pia, are found areas of more or less diffuse white or grayish-white dense thickening. Patches of grayish, gelatinous, somewhat succulent thickening, the size of a pea or larger, may be seen in any locality, more frequently on the convexity. These gelatinous patches are often located over gaps between the convolutions, and, when small, partake of the configuration.

* LeCount and Dewey: "Syphilitic Leptomeningitis," Jour. Infect. Dis., March, 1915.

In the third type of lesions are found circumscribed, homogeneous, grayish-white or yellowish masses, vaguely outlined, round, stellate, or triangular; sometimes flat or elevated in any locality of the convexity. He also found this form in the fossa of Sylvius, which is often leveled by an opaque thickening. Small nodular elevations arranged bead like in short chains or ridges were also observed.

Changes in the walls of vessels are also observed, particularly the basilar vessels. The walls show areas of infiltration and thickening. LeCount observed that rarely there was only one type of meningeal alteration; usually several were present in the same brain. If nerves lie in the region of inflammation, they become affected either by the pressure of the exudate, which may cause edema of the nerve, or by the development of a perineuritis or interstitial neuritis. Such inflammatory infiltration may be followed by more or ess extensive development of connective tissue, which results in atrophy.

It is in cases of meningitis affecting the base, particularly the region of the chiasm and the interpeduncular space, that involvement of the nerves of the eye occurs. Lesions of the membranes of the convexity, as a rule, cause changes in the optic nerve only when, by the development of large, gummatous masses, the intracranial pressure is increased and papilledema or choked disc results. This is similar to the change in the nerve that results from brain tumor. In the basilar form the furrows and depressions of the base of the brain are filled with a gelatinous, sometimes firm, bacon-like new formation, thicker in some places than in others. This new-growth is found particularly in the neighborhood of the chiasm, but may extend backward even to the pons. It may surround the different parts of the optic tract and the motor nerves of the eye. The presence of this inflammatory mass in the meninges may cause an edema of the optic nerve, expressing itself in the form of a choked disc, or there may be

a true inflammation of the nerve itself or its coverings that may extend from the brain, involving the portion within the canal and even the orbital portion of the nerve. Therefore, one may properly speak of a leptomeningitis of the orbital portion of the optic nerve.

Usually, however, it is the intracranial portion of the optic tract that is affected, and when any part of this becomes involved in the inflammation, the process may begin with an edema of the nerve or its sheath that would explain the sudden blindness in one or both eyes or in part of the field that is observed in some cases. Such attacks are often transitory, disappearing for a time, then recurring. With the continuance of the trouble there develops a perineuritis, associated with an interstitial neuritis, in which there is an infiltration of the nerve itself with round-cells and the formation of a granulation tissue which subsequently organizes and causes atrophy.

As to the frequency of lesions of the optic nerve occurring in syphilitic meningitis, valuable information is given by Uhthoff, who, in a study of 100 cases of syphilis of the central nervous system, found that choked disc or atrophy following it occurred 14 times. Optic neuritis or subsequent atrophy occurred 12 times. Simple atrophic change in the nerve occurred 14 times. In 52 of the cases the ophthalmoscope showed a normal condition of the fundus, but, of these, 5 showed homonymous and 2 temporal hemianopsia. In 3 of the cases coming to autopsy there were no disturbances of vision and no ophthalmoscopic findings, although there were distinct pathologic changes in the basal optic tracts. As to the relative frequency of "choked" disc or optic neuritis in this condition, Uhthoff has found that when choked disc occurs it is caused, in 65 per cent. of cases, by gummata of a tumor-like size, whereas in 23 per cent. the cause was basilar meningitis.

In many such cases, therefore, we will see choked disc

that cannot be distinguished clinically from that seen in intracranial neoplasm. In others there is a distinct optic neuritis, while in a number no ophthalmoscopic signs will be present. In the last-named group there may not be lacking defects in the fields as indicative of change in some part of the optic tract. Field defects vary from symmetric contraction or sector losses to hemianopsia or even complete blindness. In one eye there may be complete blindness; in the other a sector defect or hemianopsia. This variability in regard to fields is frequently a noticeable feature of such cases.

General Symptoms.—As to general symptoms, according to Oppenheim, headache is one of the most constant, but even this may be slight or entirely lacking. In some cases it is severe, often with exacerbations at night, and at times may be accompanied with vertigo or vomiting. Naturally in severe cases there might be attacks of convulsions or periods of unconsciousness. Psychic manifestations may be noticed, such as mental dullness and confusion, apathy, loss of memory, and even mania. Disturbances of speech, aphasia, apoplectiform attacks, paresthesia, hemiplegia, deafness, difficulty in swallowing are some of the numerous symptoms that have been recorded in such cases, some of which may be occasioned by the involvement of the vascular system.

But the most striking characteristic of such cases, according to Wilbrand and other observers, is the variableness, the inconstancy of the symptoms, and their sudden changes. This is also illustrated in the eye symptoms, which vary sometimes as noticeably as do the general manifestations. The suddenness of blindness in one or both eyes, and at times rather prompt recovery, the appearance and disappearance of blind areas in the field, the paralysis of motor nerves of the eye, followed by recovery, are examples of the fickleness of the process in this disease. The noticeable lack of symp-

toms of the nervous system in certain cases and the insidious nature of the trouble add to the difficulty of diagnosis when clinical history or other signs of syphilitic infection are not present. The valuable discoveries of recent times in regard to the etiology, diagnosis, and treatment of syphilis have proved of great benefit in elucidating conditions of the central nervous system that formerly presented baffling problems.

Before our diagnosis was aided by the Wassermann and Noguchi tests, and the important cytologic and chemical findings in the spinal fluid, we were compelled to rely on the clinical manifestations of the disease, which so frequently are obscure and uncertain. Not that these tests are absolute or infallible, but they have shown their value so frequently where clinical evidence was lacking that they have become indispensable adjuncts and mark an era in the diagnosis of syphilitic infection.

The prognosis as to vision is usually uncertain, but will depend partly upon the promptness of the diagnosis and the amount of damage the optic tract has suffered before effective treatment is begun. The remarkable instances of recovery of vision, in some cases complete, where the ophthalmoscopic appearances show extensive atrophic changes, indicate that the hope of cure is not to be given up even if complete blindness has supervened. Energetic treatment may save a fair degree of vision.

I should like to record the following cases which I believe to be syphilitic leptomeningitis, and which illustrate the insidious nature of the disease, the suddenness of development of the eye symptoms, and the prompt response to treatment that may sometimes follow.

CASE I.—Miss S. L., aged twenty years, was admitted to the Presbyterian Hospital August 15, 1914, with the history that on August 3d the right eyelids began to swell at the nasal side and this swelling increased so that in two days it

was impossible to open the eye. The physician who examined her observed that when the lids were separated the upper half of the visual field was blind. The following day the left one became blind, the nasal half of the field being first affected. This was preceded by an edema of the lids beginning at the nasal side, as had occurred on the right side.

These phenomena were accompanied by some headache, not intense, and by slight mental confusion, but no other marked symptoms. In the preceding May she had some trouble of the throat, which seemed to be swollen so that she had difficulty in swallowing, and her father stated that, during and after this attack, she had considerable pain in the joints and her neck was swollen. There was also some diarrhea. Her father and mother were perfectly healthy and have several healthy, well-formed children, of whom this daughter is the eldest. The mother had never had miscarriages nor did she or the father give any history or evidence of being luetic. Wassermann test of the blood of each of them was negative.

The patient herself was a well-formed girl of about 110 pounds in weight, with no history of previous illness except that mentioned and no clinical history of luetic infection. Examination showed the eyelids of both eyes so swollen that she could only open them slightly. This swelling was due partly to edema and partly to engorgement of the veins, for the nasofrontal vein on each side was considerably distended and the superficial veins of the conjunctiva were prominent. There was a slight degree of exophthalmos, but no pulsation of the eyeball over the distended veins. No bruit could be heard on auscultation.

The external appearance of the eyeballs was normal except for the distended veins. Mobility was restricted so that the abduction of the right eye was about 35 degrees, of the left about 25 degrees or 30 degrees.

The eyes could hardly be elevated above the horizontal, but movement downward was only partially affected. There was only a slight degree of convergence when she was asked to look at the end of her nose, and with this effort the pupils scarcely changed their size. The pupils were evenly and widely dilated to about 8 mm., and did not react to the strongest light. Vision of each eye was zero. Ophthal-

moscopic examination showed the media perfectly clear. Both optic discs were blurred and swollen to about 1 D., and the physiologic depression was obliterated. The retina immediately around the discs was somewhat swollen; the veins were double the normal width, engorged, and tortuous in a vertical plane for a distance of one or two disc diameters from the margin. The arteries were about normal in width, but Dr. S. L. Allen, who had referred the case, stated that the arteries had been narrower when he had examined her soon after the attack occurred. There were no hemorrhages in the retina nor were any seen at any time. Aside from the changes in and around the discs the fundus was of normal appearance in each eye. Physical examination of chest and abdomen revealed no abnormality.

Dr. Rothstein, neurologist of the Presbyterian Hospital, kindly saw the case in consultation, has examined her repeatedly, and has directed much of the treatment. I am indebted to him for his record of examinations and subsequent treatment of the case. His examination showed: right corner of mouth a little lower than the left, but movement is equal on both sides. The tongue when protruded deviates slightly to the left, and stands in a somewhat uneven plane. Right half of maxilla a little lower than the left. Restricted mobility of the eyes as mentioned above. No paralysis in arms, and muscular power of hands normal. Below the knee the sensation to sharpness is lessened, so that the patient is uncertain whether she is touched with the point or the head of a pin. Similar uncertainty on stimulation to the arms. Sensation decreased on dorsum of feet, but not above the ankles. Sensation is slightly decreased on the dorsum of the hand. Pinching the skin and calves of legs slightly painful. Babinski plus. Patellar and Achilles reflexes lively. Slight tendency to clonus on the right side. Able to walk, but is unsteady; there is a tendency to fall to the right. Spinal fluid under increased pressure; 10 c.c. of clear fluid was withdrawn. Wassermann test of blood August 21st showed weak positive. Examination of cerebrospinal fluid showed cell count 20. Nonne negative. Wassermann negative. Wassermann test of blood August 24th showed positive +. Blood count was normal. Hemoglobin normal. Tuberculin tests were negative.

Dr. Friedberg, rhinologist and laryngologist at the Presbyterian Hospital, found the nares free and all accessory sinuses clear, with the exception that the right maxillary sinus was a shade darker under transillumination. A few days later the sinus was irrigated; no pus was found, but only a few flakes of mucus.

The diagnosis was made of syphilitic basilar leptomeningitis, and treatment by inunction of mercury was given.

September 5th: Complained of roaring and ringing tinnitus with some deafness and a confused feeling in the head.

September 7th: Deafness was marked in both ears, and she could hear only a loud noise at a distance of a few feet. Was excitable and cried a great deal, and complained of great confusion and roaring in the ears. Bone conduction for sound much impaired. Both drum membranes were clear and apparently normal except for slight retraction. This distressing condition lasted for nearly a week, and then gradually her hearing was regained and within another week returned to normal. The inflammation of the optic nerves gradually subsided and was followed by atrophy, the optic discs becoming very pale. Within a week after treatment was instituted she began to perceive light with the right eye, and gradually regained a degree of qualitative vision in the inferior temporal quadrant of the right field. There was no improvement of vision in the left.

September 30th: She received 0.45 gm. neosalvarsan intravenously, and in the next month she received three more doses of 0.6 gm., 0.75 gm., 0.9 gm. in the same manner.

She was discharged October 31st very much improved as to her general condition. She was able to recognize large objects in her right inferior temporal visual field and to distinguish colors, but as there was no central vision, it was difficult to chart the field.

Roughly, her field occupied an area on the temporal side about 15 degrees above the horizontal to about 45 degrees below the horizontal plane, and extended from about 15 degrees temporally from the fovea to nearly normal limits. She returned for observation and treatment January 13, 1915. General condition was much improved, and she had gained over 20 pounds in weight. Had not been troubled with headache and her sleep was better. Her vision was

clearer, although the field had not noticeably enlarged. Colors in her field were more readily recognized, and she could even read letters about 2 cm. high several feet from the eye. The optic discs were very white. Cerebrospinal fluid was clear, with cell count of 1, a negative Nonne, and a positive Wassermann reaction. She was given two doses of neosalvarsan, 0.75 gm., increasing doses of iodid of potash, and discharged in one week. Returned to hospital again March 25th in good general condition, but with sight about as before. Spinal fluid was clear, with cell count of 3 and a negative Nonne and a negative Wassermann reaction. She received two doses of 0.9 gm. neosalvarsan during the week she remained. Entered the hospital again June 30th for observation. No change in general condition. Vision same as before. Wassermann tests of blood and spinal fluid gave negative reactions. She has not been seen since, but is reported to be in about the same condition as when last examined.

In this case the syphilitic exudate at the base not only pressed upon the chiasm and oculomotor nerve, but probably interfered with the return flow of venous blood through the cavernous sinus, thus causing the marked engorgement of the veins of the lids and eyeball. The sudden deafness can be explained by pressure on the auditory nerves by another patch of exudate, and possibly the earlier symptoms of difficult deglutition were caused in a similar way.

CASE II.—Mrs. F. K., aged forty years, consulted me first on March 9, 1916, stating that for the past month she had noticed spots dancing before the right eye, with occasional flashes of light. At times she had the sensation that she was looking through broken or splintered glass with that eye. A few days before seeing me she noticed that she could not see objects in the right inferior nasal field, and this alarmed her considerably. Her previous health had been fairly good, except that she has had several pelvic operations. During this period of failing vision she had some headache, mostly frontal and on the right side, but not severe. She has been under considerable nervous strain from taking care

of her husband, who has tabes, and an epileptic sister. Examination showed V. R. E. = 20 20 − 2; L. E. = 20/20 − 2. Right pupil was larger than the left and reacted more slowly to light and accommodation.

Perimetric examination showed left field for white normal, red and green somewhat contracted. Right field showed a marked defect in the inferior nasal quadrant and the color fields very much contracted. Tension in each eye normal; 18 mm. by tonometer. Pupils dilated evenly with homatropin. Ophthalmoscopic examination showed media clear and fundus of each eye normal. A rather large physiologic cup was present in the right optic disc. A Wassermann test

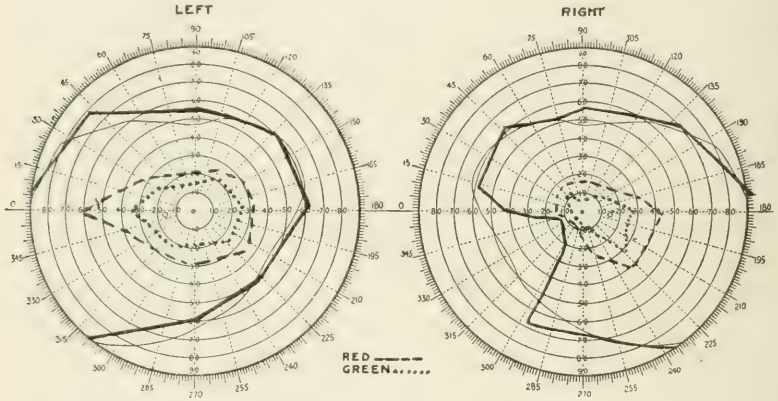
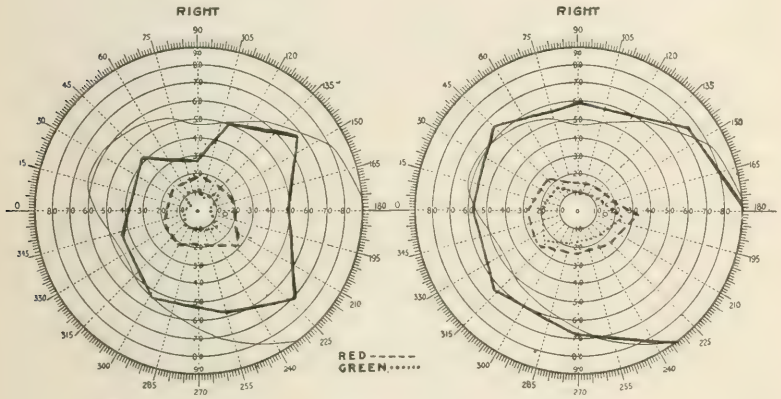


Fig. 1.—Fields, March 10, 1916. Good light. 1 cm. square object.

had been made before she was sent to me that had given a negative reaction. Four days later she called me by 'phone to tell me in alarm that she had become completely blind in the right eye. The blind area had increased each day, she said, until on March 12th she could not see with that eye. There were no spots before the left eye, but she had the same sensation of looking through broken glass that had preceded the trouble in the right eye.

I saw her the following day, and found the right pupil widely dilated and the vision of the right eye zero. There was not the faintest perception of the strongest light, nor the slightest direct response of that pupil to such light, although

its consensual reaction was prompt and almost normal. Ophthalmoscopic examination revealed no change in the optic disc nor in the fundus. The left eye had normal central vision and field about as before. I had her consult Dr. Rothstein, who, in office examination, found she had distinct decrease of sensation of touch and pain in a girdle form at the level of the mamma on the right half of the chest. Reflexes were increased. There was a dubious Babinski on both sides, and he expressed the opinion that there was localized meningitis, possibly syphilitic in character, although the clinical signs of lues were lacking and there was a report of a negative Wassermann.



Fields, right eye. Good light, 1 cm. square object.

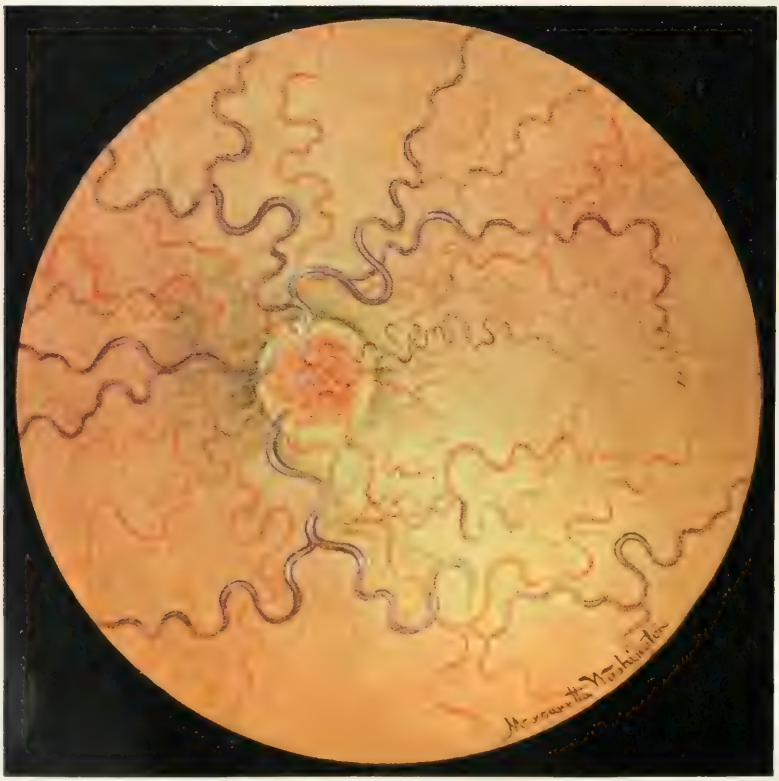
Fig. 2.—March 21, 1916. Fig. 3.—April 20, 1916.

As the case seemed urgent, she was given hypodermically 0.3 c.c. of 45 per cent. emulsion of mercury. There was considerable difficulty in persuading her to enter the hospital for further examination, and she did not go until eight days later, March 23d. In the mean time distinct improvement took place. She stated that within an hour after the injection of mercury the headache disappeared. On March 18th, four days after treatment was begun, she could distinguish hand movements close to her eye, and there was a slight reaction of the right pupil to light. Improvement then became more rapid, so that by March 21st there was vision of 20/70.

Right pupil reacted rather sluggishly. Consensual reaction much stronger than direct; with closure of the left eye the right pupil began to dilate. Fields taken at this time showed marked contraction for white and colors. On March 23d she entered the hospital. Examination of blood showed distinctly positive Wassermann. Spinal fluid was clear. Cell count 2; Nonne very weak; Wassermann weak positive. Further examinations showed that knee-jerk, ankle-jerk, elbow- and wrist-jerks and abdominal reflexes were all lively and equal on both sides. Blood count and hemoglobin index were normal. Wassermann test of blood gave a positive + reaction. The cerebrospinal fluid was clear, with cell count of 2 and a very weak Nonne test for globulin. The Wassermann test, however, was a positive +. Treatment was continued, and by March 28th right vision had improved to 20 30, where it remains at the present time. Fields taken April 20th show a return of white to normal and a contraction of red and green. No change has yet been observed in the normal appearance of the optic disc and fundus.

Patient stated on April 20th that there was a faint bluish appearance of objects viewed with the right eye, and also a faint hazy area in the field below the center, but this could not be detected by the perimetric test. On April 30th this bluish appearance had changed to a grayish one, and she was still conscious of a hazy area in the lower fields, but this could not be detected on the perimeter.

It is too early to say what the ultimate outcome of the case will be, but it illustrates well the sudden onset of partial, then total, blindness with prompt recovery that may take place in syphilitic meningitis. The blindness in this case might have been due to an edema into the nerve which was favorably affected by the prompt administration of mercury. The recognition of the syphilitic taint in a case where there had been a previous negative Wassermann, and in which clinical data were absolutely lacking, has an important bearing on the future of the case. The absence of ophthalmoscopic signs is another noticeable feature, but it may be that evidences of optic atrophy may appear later.



Cyanosis retinæ.

CYANOSIS RETINÆ ET CONJUNCTIVÆ IN CONNECTION WITH PULMONARY STENOSIS AND PATENT DUCTUS ARTERIOSUS.

ROBERT SCOTT LAMB, M.D.,
Washington, D. C.

On October 23, 1915, I was asked by Dr. Edward Hiram Reede to examine a case at the Freedman's Hospital and ascertain what the eye-grounds showed to corroborate the diagnosis of the heart condition. The case was so interesting that it seemed to warrant me in reporting it to the Society. In order to exhibit, so far as possible, the evidence which leads to the conclusion, as shown by the title of the paper, I had water-color sketches made by Miss Margaretta Washington of the external appearance of the eyes and the ophthalmoscopic findings of the fundi, and in addition arranged for the patient to come to the meeting for examination. Dr. Reede's report of his examination of the patient is as follows:

Janie Violet Hall, aged thirteen years, slight built young colored girl, moderately nourished, poorly musculatured,with swollen feet and marked cyanosis of the body. The conjunctivæ are greatly congested. Tongue is covered with a white fur through which appear very bright red papillæ; roof of mouth and the gums are markedly congested. There is visible pulsation in the suprasternal and supraclavicular region of the venous type, and pulsation of the precordial region, with a heaving apex-beat which is located in the sixth interspace, near the anterior axillary line. Both knees and ankles are swollen. The right knee is larger than the left and more painful. The nails of the fingers and toes are purple in color. The fingers are changed in shape, the ends being very bulbous, with curved nails quite clubbed in type.

On palpation no thrills nor frictions are felt. The apex-beat is fairly forcible and heaving. The vocal fremitus is not marked down the midclavicular and anterior axillary lines, but is slightly increased down the right midscapular line in its lower two-thirds. The whispered voice is not increased over the anterior aspect of the chest, but is more evident over both midscapular lines. Moist râles are audible over both lung bases behind. There is a systolic murmur heard in the pulmonic area, of greatest intensity in the second left interspace, and transmitted upward to the clavicle, blowing in quality. A second sound is audible to the left of the sternum. A second murmur is heard in the third and fourth left interspaces, systolic in time, but of quite different quality, which is transmitted to the third right interspace on the right and rather harsh in type. The percussion-note is resonant over both fronts and somewhat impaired over both bases behind. Cardiac dullness extends to the anterior axillary line on the left and beyond the sternal margin to the right. The liver and spleen are not felt. The ankles and feet do not readily pit. The history of shortness of breath and incapacitating asthenia since childhood, with cyanosis, clubbing of the fingers, and chronic venous congestion, suggests the presence of the murmur of pulmonic obstruction and tricuspid regurgitation, with venous pulsation and enlargement of the heart in the absence of signs of aortic regurgitation, aortic stenosis, or mitral regurgitation, and indicates that the lesion is probably that of pulmonary stenosis, with probable patent ductus arteriosus. The presence of a patent foramen ovale or deficient intraventricular septum cannot be determined, but the absence of a diastolic murmur is felt to be rather against their presence.

I examined the eyes, and found the palpebral conjunctiva almost purple, the bulbar conjunctiva congested, but not markedly so. The fundi were deeply pigmented, yet the distribution was neither regular nor even. There was in each eye at the fovea a bright yellow spot, while the macula was perhaps darker than normal, even for a colored girl. The retinal arteries and veins were tortuous, much dilated, yet clearly visible, as there was no evidence nor any sug-

gestion of inflammation. There was no pulsation of either arteries or veins. Vision was normal in each eye.

In addition to the water-colored sketches, I had made a radiograph of the patient's chest, and the pictures of the heart are almost identical with those of a case of similar heart lesion which came to postmortem. Other plates were made of the hands and feet, and these give an evidence of the dystrophy caused by this particular heart lesion.

In 1870 Dr. Herman Knapp first brought the subject of cyanosis retinæ before this Society, and in 1905 Dr. William Campbell Posey* read a paper and collected a total of 18 cases. Since then, so far as I have been able to ascertain, no other case has been presented to the Society. The review of the cases of this rare ocular condition has been so thoroughly done by Posey that it is unnecessary to report the case histories presented by him. Since then one case of cyanosis retinæ, with several other rare cases of different character, has been reported by Dr. Clemens Harms.† It is a full report, and the diagnosis of the heart lesion was stenosis and insufficiency of mitral, insufficiency of tricuspid, and insufficiency of aortic valves.

Five cases of heart lesions reported by Dr. T. Hondo,‡ of Tokio, refer to cyanosis of the conjunctivæ, but do not record any fundus findings.

There is a discussion of congenital heart cases by Weber and Doerner§ which records in a general way that such cases have cyanosis of conjunctivæ and fundi.

In the cases reported ‖ by Drs. Harry Wessler and Murray H. Bass, of Mt. Sinai Hospital, no record is made of an ophthalmoscopic examination.

* Trans. Amer. Ophth. Soc., May, 1905.
† Graefe's Archiv. f. Ophthal., 1914.
‡ Medizin. Klinik, March, 1908.
§ Archiv. f. klin. Med., 1911, cii, 541.
‖ Amer. Jour. Med. Sci., 1913, cxiv, 543.

One of the most interesting papers* on the subject is by Dr. Elia Baquis, on the Pathologic Anatomy and Pathogenesis of Cyanosis Retinæ, and discussion of Pathogenesis of Glaucoma. I refer to this paper for the pathogenesis of cyanosis associated with congenital heart lesion, and for explanations as to why similar cyanosis does not occur in general cyanosis from other causes.

As pointed out by Nagel, when the arteries as well as the veins are darker and broader than normal, the presence of a connection between the two sides of the heart is assured. When the darker coloration and tortuosity are limited to the veins, some other lesion of congenital origin exists which does not permit the admixture of venous with arterial blood. What, therefore, is the heart lesion in the case here reported? Probably pulmonary stenosis and patent ductus arteriosus.

DISCUSSION.

DR. T. B. HOLLOWAY, Philadelphia: In 1912 I gave the ocular findings in a case of congenital heart disease together with a résumé of the cases then on record and those that had been described since the time of Posey's report. If I mistake not, some 29 cases were analyzed. It is well known that many of these cases of congenital heart disease will exist for years before the development of cyanosis. In four or five of the insignificant number that have been recorded with ophthalmologic findings, retinal hemorrhages have been found, and it would seem that, after analyzing the cases, these extravasations occurred late in the course of the disease, and might be regarded as of more or less terminal character. Certain other conditions associated with cyanosis are capable of producing fundus pictures not unlike those seen in this condition.

Some years ago Dr. Stengel had under his observation a case of marked cyanosis due to chronic acetanilid poisoning, where a previous diagnosis had been made of a tear in the interventricular septum of the heart. Unfortunately, none of the cases of so-called enterogenous cyanosis, of which

* Graefe's Archiv. f. Ophthal., 1908.

there are a few on record, have been examined from an ocular standpoint—at least none had been up to 1912.

DR. WALTER BAER WEIDLER, New York: I would like to report a case of cyanosis of the retina associated with patulous foramen ovale. The patient was a girl, ten years of age, and at the time of birth described as a "blue baby." At the present time the child shows a great amount of bluish discoloration of the lips, tongue, and all mucous membranes that can be examined, and the finger-nails have a bluish discoloration. The child takes colds very readily and is unable to play or endure the slightest amount of exertion without undue fatigue and exhaustion.

Ophthalmoscopic examination of the eyes showed media clear, disc oval, 5 by 7 mm. long, axis 90°, scleral ring all around, with small central excavation. The veins and arteries are tremendously enlarged and tortuous, the caliber being about double the usual size. The veins and arteries are much darker in color, giving a bluish reflex. The retina is extremely granular and gives a very bluish red reflex, which is seen only in the extreme cyanosis. Vessels long, axis 90°; visual acuity is normal with proper correction. The diagnosis was cyanosis of the retina due to patulous foramen ovale.

DR. ROBERT SCOTT LAMB, Washington, D. C.: In connection with what Dr. Holloway has said, I think that we should be careful to distinguish, as far as possible, the heart lesion and its association with the fundus condition. There is a difference between the venous picture of mitral regurgitation and mitral stenosis and that of the case I have exhibited, and I think it important not to confuse congenital heart lesions generally with this type, which is distinctive and characteristic. In reference to the condition of the general system in which there is a low venous pulse, it is recognized that such a condition exists in these cases, but we must help the practitioners of internal medicine to a better understanding of the fundus picture as corroborative evidence in connection with these passive congestions due to low venous pulse.

A CASE OF PITUITARY BODY DISEASE WITH REFERENCE TO THE EFFECTS OF EARLY OPERATION.

WILLIAM EVANS BRUNER, A.M., M.D.,
Cleveland, Ohio.

There is an extensive literature upon tumors of the pituitary body or in the region of the sella turcica, so that it scarcely seems worth while to record an additional single case, and yet, because of the interesting ocular features which this patient presented at a very early stage, and because of the results of early operation, the case does seem worth reporting. We all occasionally see patients in the late stages of tumors of the pituitary body, with serious visual defects and more or less complete temporal hemianopsia or blindness of one or both eyes. Medical treatment usually accomplishes little or nothing in these cases, and it is only within a few years that surgery has been able to do anything for these unfortunates. Now, however, much can be accomplished, not only to alleviate general symptoms, but also—and this is what specially concerns us as oculists—the sight can often be preserved, and when vision is already seriously affected, much improvement can frequently be obtained by operation. It becomes the duty, therefore, of oculists to be more on the lookout for the earliest symptoms of any lesion pressing upon the chiasm, and then not to delay in sending such a patient to a surgeon competent to handle the condition.

Nothing further is needed to confirm these statements than a study of the work done by Dr. Harvey Cushing and reported conjointly by Dr. Cushing and Dr. Walker.

Without further introduction or comment, the following notes on a case of pituitary disease are presented:

Mr. X., aged forty-one years, first consulted me in March, 1909, because of some blurring recently in reading, eyeache, and tiring of the eyes after use; V. O. D. = 6/5 partly; O. S. = 6/5 partly; 0.62 p.p., O. D. 35 cm.; O. S. 36 cm. Ophthalmoscopic examination: O. D. media clear, disc round, physiologic cup, slight scleral ring out, inner edge slightly hazy; retina striated and granular, uncovering of choroid, low H. O. S., condition the same. Discs are possibly a trifle gray in the deeper layers. Under homatropin: O. D. sph. + 1.00 V. = 6/5; O. S. sph. + 1.00 V. = 6/5. He was given sph. + 1.25 in each eye for reading.

These glasses were very satisfactory, and in May, 1912 (three years after), he came again with about the same complaint, some effort in reading, and occasional slight frontal headache. Without mydriatic: O. D. +0.50 V. = 6/5; O. S. +0.50 V. = 6/5. Reading best with +1.75 added, or +2.25 in each eye. This was accordingly ordered. Ophthalmoscopic examination shows the same condition as before, a slight scleral ring in each eye, and discs possibly a trifle gray in the deeper layers. The anterior chambers were normal, tension normal, form fields normal.

In May, 1913, he came complaining of a little slowness in changing focus; V. with above glass (sph. +0.50) was 6/5 in each eye. He selected the same glass as before for reading. The fundus showed no change. He was given a pair of bifocals. A year later, in April, 1914, he came again complaining of a little difficulty and slowness in focusing: O. D. sph. +0.75 V. = 6/5; O. S. sph. +0.75 V. = 6/5. Ophthalmoscopic examination showed the same appearance as before. The left nerve is possibly a trifle more gray than the right. The focus fields were normal, but he was requested to return the next day for a careful test of his color-fields. These were found to be practically normal, except for some contraction on the temporal side in the right eye. There was also a small scotoma for red, but not for other colors, just beyond fixation out to 10 degrees; at the widest portion it extended 5 degrees or more below the horizontal line. There is no absolute scotoma in this area, but the white spot seems a trifle

more hazy. In the left eye there is a similar though larger scotoma for red, but not for other colors (Fig. 1).

The patient does not use tobacco and takes only a very occasional drink of any alcoholic beverage. At times he is annoyed with slight eczema, has chronic constipation, also hemorrhoids, and some irritability of the bladder. He occasionally has slight trouble with one ear, has had tonsillitis, though not recently, and the tonsils are rather ragged in appearance. The nose shows a slight hypertrophy of the left inferior turbinate. He considers his general health excellent, has no headache, has occasional slight, rheumatic-like

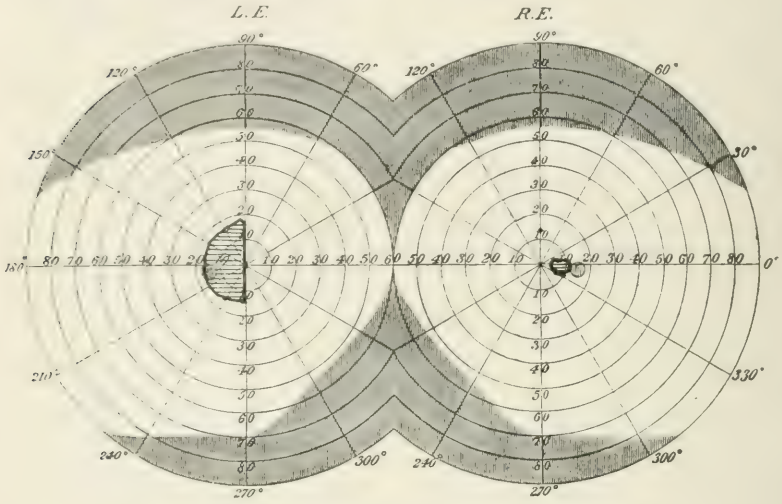


Fig. 1.—Form fields and scotoma for red.

twinges; has increased in weight 20 pounds during the past four years.

He was sent to his physician with a report of a probable growth in the sella turcica pressing upon the chiasm, and a thorough general examination was requested, and an *x*-ray of the skull advised. The general examination was negative, but, as the symptoms were so slight, the doctor did not want an *x*-ray taken as yet, lest it might alarm the patient.

In July (two and one-half months later) the patient complains that he cannot read or add up a column of figures as

rapidly as formerly: he must look directly at an object, and requires a strong light to see clearly. With glasses, V. O. D. =6/5, partly; O. S. =6/6, partly. The temporal part of each optic nerve is slightly pale, with a faint scleral ring. The fields show the color scotoma for the small red spot larger on the temporal side than formerly. He is unable to recognize it on the temporal side of the vertical line, but can tell it at once when the object is carried to the nasal side. In the area of this color blindness a small white spot (1.5 mm.) is very faint and disappears at points. In the left eye the temporal color scotoma is considerably enlarged (Fig. 2).

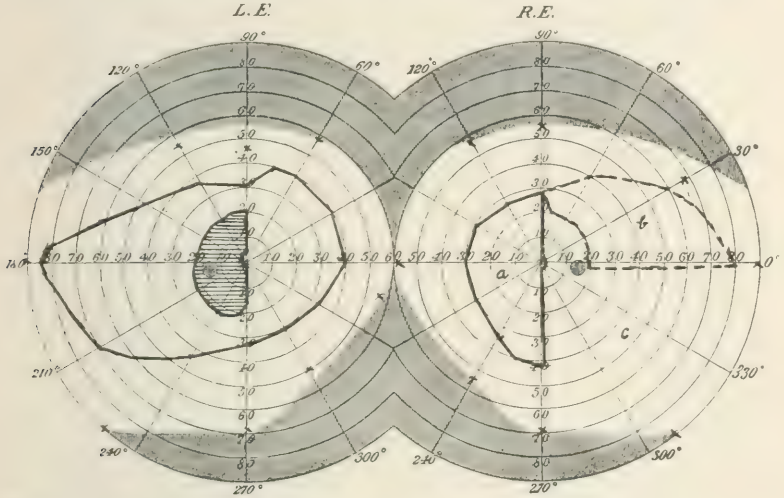


Fig. 2.—Field for red and scotoma for red. *a*, Good; *b*, faint for red; *c*, red blind.

Examination of the nose and sinuses was again advised, including an *x*-ray of the latter, and at the same time also an *x*-ray of the base of the skull, but nothing was done. In October he had not noticed any change in the eyes. With glasses, V. O. D. =6/6; O. S. =6/12. With O. D. alone he reads 0.50 print with difficulty, while with O. S. he reads only 1.75 print with effort. The outer limits of the form field are normal in each eye, but there is now a small absolute scotoma to the temporal side of fixation in each (Fig. 3).

He was now sent for an examination of the nose and sinuses

to Dr. Ingersoll, who reports these structures normal. Drs. Hill and Thomas, who took the radiographs, report "the sinuses clear, but stereoscopic plates of the head show the sella turcica to be somewhat deeper than normal at the posterior part, and there appears to be some destruction of the posterior clinoid process, suggesting a tumor of the hypophysis."

A Wassermann was made and found negative. He then went on to see Dr. Harvey Cushing, who confirmed the diagnosis of tumor of pituitary body. He advised against any immediate operation, but favored watching for several

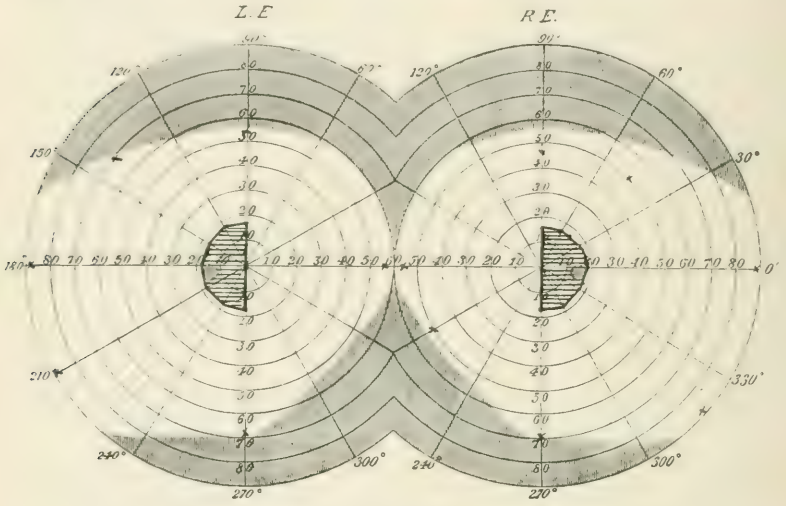


Fig. 3.—Scotoma for red and partial for small white spot.

months, and, should the symptoms increase, he would then operate to preserve his eyesight. He advised a trial of pituitary extract in the meantime.

In December his condition was about the same. The temporal half of each nerve was rather pale, especially the left. January, 1915, he again saw Dr. Cushing, who then advised operation. Dr. Walker at this date corroborates the color defects, and reports in addition "characteristic bitemporal form defects found when the disc diameter was decreased below 1 mm."

The following month the patient returned to Boston and was operated upon by Dr. Cushing. Dr. Cushing reports the operation a transsphenoidal one. He found "a particularly large sphenoidal cell which contained a median division. The exposure of the floor of the sella was particularly satisfactory. It had a rather broad exposure transversely in comparison with the usual narrow transverse exposure. The mucous membrane was removed from the cells, and the floor of the sella was then rongeured away, giving a wide exposure. In pricking the dura a yellow, soft struma began to extrude. An opening the size of the thumb-nail was made in the dura, and many fragments of the struma were removed." Recovery was prompt and satisfactory.

He reported at the office April 7, 1915, about six or seven weeks after the operation; was feeling finely. Could see better than before, and no longer had any trouble with figures. With his glasses, V. O. D. = 6/5; O. S. = 6/5. He readily reads 0.50 print with either eye. Ophthalmoscopic examination shows the nerves about the same; temporal edge of right disc slightly pale, the left more pale, with scleral ring also more marked. The absolute scotomas to the temporal side of fixation have disappeared, while only a small color scotoma at this location remains in each eye.

In October, 1915, eight months after the operation, V. O. D. = 6/5; O. S. = 6/5, but slightly hazy; reads 0.50 readily with each eye. The fundus shows no change; the form fields are normal in each. There is still a small scotoma for red, but not for blue or green, between fixation and the normal blind spot. He is feeling well, and the eyes are giving him no trouble. On March 29, 1916, he reports that he is getting along finely. He has recently seen Dr. Walker and Dr. Cushing, and was told by them that the eyes are the same or slightly better than last fall. The form field is normal on the temporal side in each eye, and there is no scotoma near fixation, except for color as before. He is having no difficulty whatever in using his eyes.

DISCUSSION.

DR. G. E. DE SCHWEINITZ, Philadelphia: I am satisfied that a symptom to which I called attention in 1912 is an important one in the study of the ocular signs of pituitary

body disease; namely, a form of blurred vision of which the patient is definitely conscious, and which antedates by a long period of time the amblyopia which is associated with ophthalmoscopic nerve-head changes, or any form of scotoma, or any visual field defect (searched for by the usual methods), or any failure to read normally the ordinary test-types. This period of time may last for months, even for years. In one patient it continued almost for two years before change in the color of the disc was noticeable. It is a symptom difficult to describe. It may be unilateral. The patient reads the letters correctly, but is conscious of a definite mist, often in the central area. Evidently this mist interprets a beginning physiologic block to light impulses. I have discussed this symptom with Dr. Cushing, and also with Mr. J. Herbert Parsons, who had also observed it; that is, observed a period of amblyopia preceding the development of scotoma.

I am not so pessimistic in regard to the value of organotherapy as Dr. Bruner appears to be, if I understood him correctly. I have placed upon record the case history of one patient with typical symptoms of pituitary body disease, confirmed in the usual manner, who was totally blind for a long period of time,—twelve days in one eye and several weeks in the other,—who regained full visual acuteness after a prolonged course of thyroid extract; also, however, mercury was administered in the form of inunctions. But I have recently completed the observation of a patient who developed the typical visual phenomena and *x*-ray symptoms of pituitary body disease, who declined operation although it was urged, and who took persistently for many months pituitary body extract, with the result that the visual fields were completely restored and the vision, which at its lowest ebb was 4/150, is now 6/5 in the right eye and 6/12 in the left, and all headaches and other symptoms have disappeared. She took three to five grains three times a day of pituitary body extract for many months.

DR. BRUNER: I would simply say that this patient was given pituitary extract without any noticeable change in the symptoms.

HIGH HYPEROPIA FROM A CLINICIAN'S STANDPOINT.

HOWARD F. HANSELL, M.D.,
Philadelphia.

A clinical analysis of 200 cases of hyperopia of three diopters and higher, taken alphabetically from the case histories, was undertaken for the purpose of learning the reasons that obtained in preventing each and every case from gaining relief from asthenopia when the estimation of the refraction was accurately made.

No especial difficulty in the refraction work presents itself in the absence of disease or irregularity of the refracting surfaces. Vision of full acuity cannot always be obtained, but vision less than 6 6 is not incompatible with a perfect correction, for reasons that readily suggest themselves.

The hyperopia of aphakia, because of the loss of accommodation and the thereby acquired abeyance of the relation of the intraocular and extraocular muscles, should be excluded, except for comparison.

Each case in which either a part or all of the hyperopia is latent presents a problem by itself. It is an easy matter to follow the usual routine of paralyzing the accommodation and determining the combination of sphere and cylinder which will give the best vision, but it is quite another matter to order and to have correctly made the pair of lenses which will relieve the asthenopia.

Theoretically, the combination which neutralizes the entire optical defect when the glass is held at the proper distance from the cornea should be worn. This, as we all know, is, as a rule, impracticable. The amount to be deducted from the

full correction varies rather with the ocular requirements of each case than with the degree of hyperopia. If regulated by the latter, as seems to be the rule, the deduction is in direct proportion to the hyperopia. A few patients among those of the extremes of hyperopia may wear full correction with comfort. In the majority deductions must be made for the sake both of satisfactory vision in the distance and the avoidance of sudden change of relations between accommodation and convergence.

The obstacles to the relief of asthenopia, after the error of refraction has been accurately determined, are to be sought for in the relative convergence and accommodation, or in the construction or fitting of the lenses to the eyes individually and as a pair. Hess states the proposition simply and assumes that, by following his suggestion, no difficulties will be encountered. He says: "Therefore, one chooses deliberately convex glasses of the strength that the ciliary muscle shall only be depended on or have work enough to see clearly in distance and near without asthenopia." The whole problem is thus briefly summarized and dismissed and appears to be, from Hess's standpoint, extremely simple. That it is complex and difficult we know full well.

Disruption early in life of the normal relation of accommodation and convergence—one diopter to one meter angle—may not be permanent, and balance may be restored by suitable glasses. For example, internal strabismus, without amblyopia, is often corrected without operation. But in adult life the habit of years is less readily overcome. The ciliary muscle has gained in power, or the lens curvature, while under the influence of the chronic contraction of the muscle, is increased, thereby diminishing the hyperopia for all waking moments. One can hardly conceive of the independent action beyond the relative range of the accommodation, and even outside the relative range, unless artificially produced, the accommodation demands conjoined and pro-

portionate convergence. Under full cycloplegia the power of convergence is unaffected. The adaptation of a glass that will restore vision to 20/20 presents no difficulty in the absence of ocular disease, but the comfortable wearing of that glass after cycloplegia has passed away is quite otherwise. Reckoning must be had with both the restored accommodation and with the new relation incident to the glasses, between convergence and accommodation. Some patients unconsciously and immediately adjust themselves to the new conditions. Others never do.

Mrs. F., aged thirty-eight years, has been under my observation for fifteen years. She suffered with nearly constant headache, the only apparent cause for which was hyperopia of about three diopters. The degree of hyperopia and intensity of asthenopia varied from time to time, and new corrections were given yearly. She had absolute freedom from headache only when both eyes were under the influence of belladonna. No combination of spheric glasses, or full or partial correction, or the constant use of the glass, or its use only in near, availed. No two consecutive examinations resulted in the same formula. There was always an uncertainty and indecision during the testing when the cylinder axis was shifted a few degrees or when the strength of the sphere was slightly increased or diminished. Retinoscopy showed a decided difference between the refraction of the pupillary area and the periphery of the cornea. Therefore, examination with dilated pupil must necessarily be followed by post-mydriatic refraction. Notwithstanding the overaction of accommodation this patient had a low grade of exophoria for distance and a higher grade for near. She was one of those patients, not infrequently met with, who wander from one oculist to another and who never get relief in spite of most careful refraction, because of an incorrectable disturbance of the relation of accommodation and convergence, probably dependent on accommodative strain during school life.

The size of the pupil is a factor that should not be ignored. A correction that is not full, or nearly full, favors contraction

of the pupil and consequently diminishes the functioning area of the cornea, and makes the demand for a strong glass even less imperative.

Differences in the power and range of accommodation in the two eyes, to which Duane has recently called attention, if unsuspected and unsought for, will prevent full relief. The difference may depend upon less hyperopia in one eye or be independent of the refraction. At the time the post-mydriatic examination is made, when the refraction of the two eyes is known, the near-point of each eye is to be determined and the glasses ordered modified by the findings.

Mrs. Y., aged twenty-five years, has never worn glasses. Complains of asthenopia and intense headache. Vision equals 20/20. Hyperopia of 3.50 D. orthophoria. She was entirely relieved of all her symptoms by 50 D. less than full correction. This patient's immediate adaptation to the new conditions controverts all our theories. The unconscious constant use of 3.50 D. of accommodation produced no vertical or horizontal muscular imbalance. The eyes immediately adapted themselves to the revolution in their use, and the muscular coördination was unmodified.

Numerous cases of this kind may be unearthed by a search through our records. In some the hyperopia is associated with a low degree of astigmatism, and in others it is uncomplicated. The ciliary muscle is probably not really hypertrophied, and the excess of convergence, stimulated for years by the accommodation, has been within the limits of the normal relation. The correction immediately removes the excessive stimulation to the third nerve which apparently had been the only abnormality traceable to the hyperopia.

The explanation of such inconsistencies in two persons of the same age, sex, and methods of living, with practically the same optical defects, may be temperamental, a factor frequently overlooked in the oculist's office and impossible to foretell.

In the higher grades of hyperopia, 6 D. and upward, relief from asthenopia is readily obtained by wearing approximately full correction. The patient feels the necessity for a glass. His eyes are useless for near work and for accurate vision without it, and in spite of annoyance he is willing to wear the glass because of the better vision until he finds it essential for both comfort and vision. The same statement applies to high hyperopic astigmatism. The adaptation to the full correcting glass, provided the axis is accurate, is immediate. There may be accommodation and convergence anomalies, as in simple hyperopia of equal degree, but if present, they are lost sight of in view of the immediately recognized advantages secured by wearing the glass.

Mrs. B., aged twenty-seven years, had daily headache, dizziness, vomiting. She wore for eight years with entire relief R. E. sph. +4 ⊂ cyl. +3 ax. 75°; L. E. sph. +3 ⊂ cyl. +4 ax. 97½°—0.50 D. less than full correction. The symptoms returned, to be again relieved by a slightly stronger glass, and 5° change in the axes of the cylinders. The muscles remained in balance.

Mr. C., astigmatism of 4 D. and 4.50 D. and esophoria of 7°; had no asthenopia whatever after commencing to wear full correction for astigmatism.

K., aged eleven, never wore glasses. R.E. sph. +11; L. E. sph. +11, full correction, became immediately indispensable.

One is inclined to charge the unrelieved asthenopia to an impairment of general health, and often correctly. Oncoming illness, chronic invalidism, or convalescence may neutralize the most accurate correction of refraction errors. Idiosyncrasy, disposition, unwillingness to accept new thoughts or impressions that do not originate in oneself, must be taken into account.

The psychology of the ocular radical revolution brought about by the beginning of the wearing of glasses for the correction of high defects presents an obstacle not often ser-

iously considered. Routine refraction work, however careful, is sometimes unavailing unless strengthened by a feeling of confidence on the part of both physician and patient that the glass prescribed is the right and only treatment. Persistent wearing of the glass will prove that the confidence has not been misplaced. No other remedy can be efficacious, and the sooner the patient admits it and yields, the sooner is he relieved. A patient said to me that if she had selected the most unsuitable pair of glasses in a store they couldn't be worse than those I ordered. She had consulted me against her will. In a few months she returned, voluntarily confessed that she had worn the glasses constantly, and the eyes were entirely comfortable. Some psychic influence dominated her thought and action irrespective of the character of the glasses ordered. Such persons are difficult to deal with and tax to the utmost the refractionist's easily exhausted patience. Only a consciousness that the patient's judgment is at fault or his mind temporarily unhinged may carry the treatment to a successful conclusion.

The oculist's best work, the result of the combined function of all his mentalities, may be neutralized by inaccurate grinding of the lenses, involving both the two curved surfaces and the optical center or the maladjustment of the lenses before the eyes. The creation of one-half or higher degree of hyperphoria by the glass will be a source of discomfort. Such an artificial muscular imbalance is not infrequent. It may be detected by a simple and easily carried out method. It is assumed that the muscular status before refraction and with the correction is known. The Maddox rod or other tests for heterophoria may be applied to the optician's completed glasses when the patient brings them for inspection. Variation from the previous findings indicate prismatic action of the lenses. An apparently perfectly fitting nosepiece may be the source of trouble by compressing the skin and vessels or by dragging the median end of the lower lid

away from the ball, an unsuspected obstacle to the relief of lacrimation, asthenopia, and the comfortable wearing of glasses.

Asthenopia, in cases in which one eye has been destroyed, is promptly relieved by accurate correction, principally because in such patients there is no convergence and accommodation relationship, and the power and range of accommodation of one eye only to be considered. A full, or nearly full, correction may be worn without loss of visual acuity. For instance, Miss L., aged twenty-seven, has 20/15, partly, with sph. +3.25 ⌓ cyl. +1 ax. 75°, full correction, which she wears with comfort.

The obstacles to the perfect relief of asthenopia most frequently encountered are incomplete adjustability of the convergence to the altered stimulation to the ciliary muscles or association of accommodation; differences in the accommodation of the two eyes; the mental attitude of the patient toward glasses or the oculist, ill health, and the imperfect work of the optician.

UNUSUAL COMPLICATIONS IN A CASE OF GLAUCOMA AFTER ATROPIN.

T. Y. SUTPHEN, M.D.,
Newark, N. J.

Unusual clinical cases are, as a rule, instructive and worth recording, and as this one was new to me after many years of special work, I am giving the main facts as briefly as possible.

Mrs. F., Russian by birth, a vigorous, healthy woman, aged thirty-eight years, presented herself at the clinic November 2, 1914, with asthenopic symptoms. She gave no history of former illness, had had children, but not for several years, but had missed her last menstruation—an unusual circumstance. V. = 20/20, each eye. A solution of atropin,

one-half per cent., was prescribed as a cycloplegic. When she returned, two days later, V. R. E. = 20/30, with sph. +1.25 = 20/20; L. E. = 20/30, with sph. +0.50 ⌒ cyl. +0.25 axis 150° = 20/20. Her next visit was on November 11th, when I saw her for the first time. She then complained of great dimness of vision. There was slight pericorneal injection, pupils widely dilated, the right cornea steamy, tension 30. V. = 20/40 each eye. Warm compresses locally were ordered, and a 2 per cent. solution of pilocarpin. November 13th both pupils were contracted to normal size; tension normal, as judged by palpation; ophthalmoscopic examination negative; no cupping of either disc. November 15th V. = 20/20, each eye. One week later she came again to the clinic, when she was seen by one of the assistants, a man of experience, who felt sure there was a mild iritis, the pupils being contracted and slight synechia in the left. Atropin was again prescribed. November 28th the patient returned with a decided glaucomatous condition in both eyes, pupils dilated, anterior chamber shallow, cornea steamy, tension high, and vision reduced to fingers at 10 feet. Eserin solution (gr. ¼ to ℥j), together with moist heat, was prescribed locally, and sodium salicylate internally. Two days later, being no better, she was admitted to the hospital and a paracentesis of each cornea was made. Twenty-four hours later, December 4th, as the patient's condition was still bad, an iridectomy was performed upon each eye. Hemorrhage followed the corneal incision in both eyes, and made the operation difficult. The tension was reduced at once. Upon removal of the dressings for the first time the left coloboma was found correct, but in the right eye the iris was seen to be entangled in the angles of the corneal incision. The tension in that eye was slightly greater than that in the left, which was normal apparently. The patient did well until December 9th, when the left cornea became milky—not especially like striped keratitis, but looking more like a general infiltration of the corneal layers. To my dismay this appearance steadily increased for several days, and I was anticipating a sloughing of the cornea, with all its sequelæ, when the trouble began to mend under rather strenuous applications of hot compresses, and continued to improve rapidly, the cornea being again clear in about a week. As the tension of the right eye still was

greater than it should be, a broad corneal incision was made with a Graefe knife, completely severing the columns of the entangled iris, and the tension in that eye was reduced at once and remained so. The patient was allowed to leave the hospital for the holidays, reporting at the clinic occasionally, and continuing the use of eserin, as the pupillary spaces were larger than they should have been, although the tension was good. On January 10th the patient reported that the evening before, while making a great effort to see some small object, she was startled by a sudden and severe pain in her left eye—her better eye. At the lower nasal sclerocorneal border, some distance away from the corneal scar, was seen a dark protrusion the size of a buckshot, evidently a sclerocorneal rupture with prolapse of the iris at its periphery. There was no history of any injury which might cause such a condition. Although a pressure bandage was applied, this little swelling slowly increased for ten days until it was the size of a large pea, oblong in shape, its greater length corresponding to the border of the cornea. A free section was then made through the protrusion, which collapsed as the aqueous escaped. A pressure-bandage was again applied, and this performance was repeated several times at intervals of two or three days. The swelling gradually grew smaller, and in about three weeks was completely gone, the surface being level with the surrounding cornea. The patient reported occasionally at the clinic, still continuing the use of eserin. A test made April 20, 1915, showed the vision of the right eye to be 20 100, with correction of an enormous corneal astigmatism, while in the left eye, the one having had the sclerocorneal rupture, she saw 20/30 with correction. There were no further signs of glaucoma, and tension was normal in both eyes. April 26th V. R. E. = 20 100, with cyl. +4D. axis 120°; L. E. = 20/20 with cyl. +2.50 axis 150° ⊂ cyl. −3.50 axis 60°. May 18th V. R. E. = 20 100; L. E. = 20 20+ with above correction. When I saw the patient in November, 1915, just before she left for South America, and one year after the beginning of her trouble, the vision was still 20/20+ in the left eye, and she could read the smallest type readily.

This case is reported chiefly on account of the spontaneous sclerocorneal rupture and prolapse of iris, and the excellence

of vision after treatment by repeated paracentesis. It also emphasizes the great mistake of neglecting to relieve iris entanglements, especially in glaucoma, as shown in the other eye. The immediate improvement in tension, when the pillars of the entangled iris were cut across, was conclusive, to my mind, that this should have been done earlier. The danger of the use of atropin in certain cases under forty years of age is also exemplified, but I do feel that no other drug will so completely and surely give us absolute paralysis of accommodation, especially in childhood. Why this rupture occurred I am unable to say, unless it be that there was a peculiar lack of resiliency in the cornea, as indicated by the unusual infiltration into the layers of that membrane following the iridectomy. I might add that the woman menstruated regularly during the entire time she was under observation.

As I look back upon the history of this case it seems a warning against temporizing in such a serious condition as acute glaucoma, and had a Lagrange or a similar operation been made early, no doubt good vision would have been preserved in both eyes.

A CASE OF BOTULISM.

WALTER B. LANCASTER, M.D.,
Boston, Mass.

Botulism presents a good many points of interest to the ophthalmologist as well as the bacteriologist, pathologist, neurologist, and also the housewife and manufacturer of preserved foods. It is probably not so rare as it seems, because it is not always recognized. As botulism has never been made the subject of any communication to this Society, the report of a case in which this seemed the most probable diagnosis may be justified on the score of novelty, if nothing else.

Miss X., aged twenty-three years, has a family and personal history which shows nothing bearing on the present illness. For some weeks she had not been feeling as well as usual and was overtired. On August 9, 1915, she went to bed at ten, feeling as usual. Two hours later she woke with diplopia and pain in the eyes. The next morning she went out on an errand, hoping to work it off, but was hardly able to get home alone on account of the vertigo associated with the diplopia.

When first seen by me on the second day the striking features presented were: Total ophthalmoplegia except of the sphincter of the iris; that is, there was complete ptosis, and on raising the lids the eyes were found to be immobile; pupils not dilated and react slightly to light; distant vision apparently good, but needs convex lenses to read fine print at the near; owing to the difficulty in fixation it is not easy to decide whether vision is diminished—it certainly is not markedly diminished, though the patient complains that she cannot see well. Nothing abnormal found in the fundi, which were sufficiently well seen to detect any well-defined changes, but not sufficiently well seen to eliminate positively slight pathologic conditions. (Later examinations under more favorable conditions confirmed the first finding.) There was distressing nausea, greatly exaggerated by the slightest movement. The patient has the room kept dark, and dreads the light, although it causes no pain, probably because in the dark the visual disturbance is more in abeyance and so the vertigo and nausea are diminished. There is moderate dysphagia; speech is somewhat difficult (tongue); no excess of mucus or disturbance of secretion in the mouth or throat; bowels loose as a result of vigorous treatment with calomel, salts, and enemata; has vomited many times. No facial paralysis detected, though later there was left facial paresis which lasted about six weeks. Hands show tremor; there is general muscular weakness, especially marked in the neck, so that the head cannot be raised from the pillow. Patient is somnolent, sleeps eighteen hours a day, otherwise mental condition is clear and good. Pulse, 110; respiration, 25; temperature, 102° F. Patient seriously ill and a good deal exhausted by even the slight examination made. Additional data supplied by Dr. Marble, who had charge of the case, and to whom I am greatly indebted for permission to

report it. Physical examination: Nothing abnormal about mouth or throat except enlarged tonsils, which have frequently been inflamed, but at present show no active condition; no enlarged glands; heart and lungs normal except for old healed trouble at left apex; abdomen negative except for slight tenderness over the gall-bladder and evidence of fecal accumulation in the colon, both of which passed off in a day or two following free evacuation of the bowels; urine, no abnormal constituents; blood-pressure, 120; blood count, 7000 white, 4,500,000 red. Aspect of the case is that of severe toxemia. von Pirquet negative; Wassermann negative.

No associated cases came to our notice, nor could the source of the poison be traced, as the patient for several days had been eating at various restaurants and on several occasions had partaken of chicken salad and similar dishes.

Subsequent course: The fever subsided by lysis at the end of ten days, and thereafter for over a month ran a course ranging between 98° and 99.5°. The vomiting lasted only for one day, but the nausea continued for weeks, and even after months a sudden movement in the wrong direction caused nausea and vertigo. Somnolence lasted for twelve days. Appetite and strength slowly returned.

On September 18th, six weeks after the first symptoms, she was sitting up and with help walked across the room to meet me; both weakness and incoördination were in evidence. With the aid of the frontales the lids could be raised enough to see some without using the finger to lift the lid, as had been necessary when last seen. The eyes were not wholly immobile, there being some power of convergence. With a sph. +4 lens she read the finest print over a range from 25 cm. to 14 cm., equal to three diopters of accommodation.

October 22d, first seen at my office. V. O. D. cyl. + 0.75 ax. 20° = 6/8; O. S. cyl. + 0.75 ax. 175° = 6/10. Right eye moves 2 mm. in the horizontal meridian; left eye 3 to 4 mm.; no perceptible vertical movement.

The ptosis diminished slowly but surely, and there was a similar slow gain in the movements of the eyeballs, especially in the horizontal plane. Thus, in November prisms of 6° base in were ordered for each eye, to facilitate single vision,

and were worn with comfort. In December they were reduced to 4° each eye; in January to 3°; in February, to 0. In the mean time the vertical deviation, not over ½° in October, had increased so that in February 3° were required to correct it, and in March 4° were worn, the right hyperphoria reaching 6° at one time.

During all this time control of the other muscles was improving. Writing was affected only during the acute stage, so with voice and swallowing these rapidly improved and became normal during the first six to eight weeks. Piano playing, which showed loss of control rather than weakness of hands and fingers, became normal about the first of March. Weakness of the legs, which showed itself when trying to get up from the floor, is still present, though much improved. Lack of control of the head is noticeable only on fine tests. When fatigued, the head shows slight unsteadiness, and, as the eyes are unable to make the compensatory opposite movements promptly enough to preserve constant fixation, the vision appears diminished. If the head is steadied in some way, the eyes are then able to fix and the vision is restored to its usual standard.

The diagnosis in this case was based entirely on the clinical symptoms. The source of the poison was not traced, and therefore there is no proof that the diagnosis is correct. There are not many other possibilities to be considered in making a differential diagnosis. It is evident that we have to do with an acute paralysis of the muscles of the eye, throat, and various other parts of the body. The very rapid onset, the absence of sensory symptoms, and the absence of gastro-intestinal symptoms are striking features. In making my diagnosis I simply went over Uhthoff's list of the possible causes of ophthalmoplegia based on a very exhaustive search of the literature covering all reported cases. The acute and subacute forms of ophthalmoplegia alone needed to be considered. These are classified under intoxications, infections, trauma, and unknown. Under intoxications are listed:

Alcohol (acute hemorrhagic polioencephalitis superior (Wernicke)).
Lead.
Botulism.
Carbon monoxid.
Nicotin.
Snake-bite.

The only one of these to be for a moment considered is botulism, unless we recognize the first as not always alcoholic, which is probably true. Under infections are listed:

Diphtheria.
Influenza.
Measles and other exanthemata.
Miliary tuberculosis.
Exposure to cold.
Syphilis.

For a while tuberculosis was adopted by one of the consultants as the most probable diagnosis, but it was early given up. There is no evidence whatever of the existence in our case of any of the other infections.

Trauma may easily be excluded, and there remains the group of cases of unknown etiology. It is possible that ours should be placed here. Hysteria was early considered, but early abandoned as a possible diagnosis.

The Place of Botulism Among the Bacterial Food Poisonings.—There are three types of bacterial food poisoning. The first is due to eating the meat of diseased animals, the second to putrefied meat, the third is botulism. Ptomain poisoning is no longer recognized as a common cause of food poisoning, though still most popular with the laity and not a few medical men.

The first, caused by eating the meat of diseased animals, is the most common. It is due to B. enteritidis, B. paratyphi B, and related forms, which infect the animal during life, but do not alter the appearance, smell, taste, or consistency

of the meat, which is therefore readily eaten. It is easy to see that when the meat is made into sausage, where liver, spleen, and other organs in which bacteria abound are all ground up together, harm is more likely to follow. The symptoms are due to the toxic metabolic products of the growth of the bacilli in the meat, and also to their activity after reaching the intestine. Thus, the illness may be very acute, due to the toxins already present in the meat and rapidly following its ingestion, or it may develop more slowly, like a true infection resembling a paratyphoid fever, with which it is probably often identical. One readily understands how, if the animal was suffering from diarrhea before being slaughtered, meat from the carcass, especially sausages, may carry the offending organism to the careless or unconscious eater. Sound meat is easily infected, and bacilli carriers who handle the meat, either in the kitchen or elsewhere, are an obvious source of danger. Cooking destroys the infecting bacteria, but does not destroy the toxins, so that the severest cases follow eating the raw meat, but poisoning may occur after eating cooked meat. The symptoms are those of a gastro-enteritis, often with skin lesions. Diagnosis is difficult; from 2 to 5 per cent. of the cases are fatal.

The second group is due to decomposed meat, which may have been perfectly sound and healthy when slaughtered. Not all decomposed meat is poisonous—only when the decomposition is caused by certain organisms or perhaps under certain favorable conditions. Meat that is more or less decomposed is eaten every day with impunity. For example, game or other meat that has hung too long, various kinds of putrid fish, and cheese of various kinds. In all these a great variety of organisms are found which seem to be harmless or relatively so. The organisms most frequently found in the cases where decomposed meat has caused poisoning are B. proteus and B. coli. The meat contains toxic products produced by the growth of the bacillus, and the bacillus

may continue to grow and produce toxin after it has entered the intestine of the victim. Cooking does not easily destroy the toxin. The symptoms are gastro-intestinal; the course is rapid, usually without fever. Deaths are infrequent.

The third form of meat poisoning is botulism. The organism, B. botulinus, is a large anaërobic bacillus allied to the tetanus bacillus. It does not grow well at the body temperature, and all its mischief is done outside the human body, *i. e.*, it is never a parasite. It is said to be the only pathogenic microörganism that grows best at room temperature, not body temperature. Although it forms spores, these are fortunately not very resistant. Heating to 180° F. for one-half to one hour is fatal to them.

B. botulinus is an obligate anaërobe, and has most often been met with in sausages of the large type. (The name botulism is from the Latin for sausage, and the synonym, allantiasis, is from the Greek for sausage.) It is also found in ham, canned foods, especially beans, usually home made, in pickled fish or meat where the brine is too weak—10 per cent. salt solution will prevent its growth. While an obligate anaërobe when grown by itself, it can grow symbiotically with ordinary putrefying organisms under apparently aërobic conditions, the putrefying organism protecting it from the oxygen. Its growth in suitable media is associated with the liberation of gas, so that sealed cans in which it has grown show bulging ends—are "blown," as it is called; bubbles of gas also may be found in the liquid. The odor associated with the growth of the B. botulinus is characteristic; it is not putrid or fetid, but rancid, strongly resembling butyric acid. The toxin produced by its growth is intensely poisonous, ranking in the class with tetanotoxin. An antitoxic serum can be produced in a similar way as in the case of tetanus and diphtheria, and this is kept on hand by the government in some cities for use when needed. Unlike most bacterial toxins, botulin can enter the system by the

gastro-intestinal tract without being destroyed, although it takes a larger dose than when administered subcutaneously. Like tetanotoxin, botulin has a strong affinity for cerebro-spinal tissue, especially gray matter. If 0.1 c.c. of bouillon culture is mixed with one gram of brain tissue rubbed up with normal salt solution, the resulting mixture is several times less toxic than a similar dilution with indifferent material. That this is not due to any destruction of the toxin, but only to its union with the tissue, is shown by the fact that simply drying will break up this union and restore the toxicity. Like tetanotoxin, it is slow in being absorbed and uniting with the tissues for which it has an affinity, and which it injures, so that there is always a considerable interval between the administration of the toxin and the first symptoms—usually one-half to one day, sometimes longer or shorter. Abdominal pain is rare, but the stomach and bowels show in many cases a marked atony, which makes cathartics and emetics ineffectual, and may be one factor in the prolonged period of incubation, since absorption is slower. Hence the importance of washing out the stomach even if the patient is first seen several days after the ingestion of the poison. The chemical character of the media on which B. botulinus is grown makes a good deal of difference in the resulting toxin. This may explain why different epidemics show differences in symptoms and severity. Also, there is evidence that different strains of B. botulinus occur. Thus, the antitoxin prepared from the original strain of B. botulinus isolated by van Ermengem was found by Leuchs not to act on the toxin from the Darmstadt strain isolated from some canned beans, and, vice versa, the Darmstadt strain antitoxin had no effect on the toxin from the van Ermengem strain.

The pathology and pathologic anatomy of botulism have not been satisfactorily elucidated. Soon after the organism was discovered by van Ermengem in 1895 Marinesco studied

the pathologic anatomy of experimental botulism. He was followed by Kempner and Pollack, Ossipoff, Römer, and Stein. van Ermengem sums up the findings of these investigators as follows: it produces a cloudy, fatty degeneration of endothelium, secretory cells of certain glands, striated muscle-fibers, and especially cells of the anterior horns of the cord and its prolongation upward through the medulla and pons as far as the nuclei of the third nerve. These lesions are absent for the most part from the brain, and entirely from the cerebral and spinal nerves. All these investigators agree that the motor ganglion-cells are affected primarily by the toxin, resulting in a degeneration of the Nissl bodies. It is because the toxin has a specific affinity for certain cell groups that we get so sharply defined a clinical picture. Wassermann would explain this on the basis of Ehrlich theories as due to the fact that the haptophore groups of the toxin molecule have a special affinity for the receptors of these cells.

Now Wilbur and Ophüls and Dickson, all of Stanford University, have recently studied the lesions in some fatal cases of botulism and in experimental botulism in animals. They deny the validity of the results reported by the European investigators, and claim that the primary lesions are in the blood-vessels, viz., thrombosis and hemorrhage. As a result of the circulatory disturbances thus produced we get changes in the nerve-cells. They are still carrying on their researches, and one must suspend final judgment. So far their statements and evidence furnished are not entirely convincing. In attempting to decide between these rival theories one naturally turns to other diseases with allied pathology, such as tetanus, diphtheria, rabies, acute anterior poliomyelitis and polioencephalitis, cerebral syphilis.

In favor of the contentions of the California investigators we may cite the change of view that has occurred in the case of acute anterior poliomyelitis. It was taught by Charcot, and pretty generally agreed, that the primary lesion was a

degeneration of the motor nerve-cells of the anterior horns of the cord due to a toxin, but it has been shown that this is not the case. The germ itself reaches the spinal cord and the lesions are inflammatory. So it was thought that the cerebral lesions produced by syphilis in cases of general paralysis were due to a toxin, but it has been proved that spirochetes are present, and the nature of the lesion is probably an inflammation. The nature of the lesions in tetanus is not known. In diphtheria we have conflicting claims. There seems to be good evidence of changes in the peripheral nerves and also in the nerve-cells; the latter may be secondary to the former. Thus, the evidence is conflicting and the pathologic anatomy of botulism cannot be regarded as settled.

The clinical symptoms of botulism present a sharp contrast to other forms of bacterial meat poisoning. Instead of gastro-intestinal symptoms, we have the central nervous system affected, and it is only the efferent nerves, viz., motor and secretory, that are involved, though I think that the secretory symptoms are due to the direct action of the toxin on the glandular epithelium, rather than on the nerves.

Usually the eyes are the first to be affected, and in mild cases the paralysis may be limited to the eyes. In more severe cases the lower motor cell groups in the bulbar region and in the cord are involved, but none of the sensory nerve-centers. The only exception to this is the complete loss of temperature sense on the whole right side of the body, below the fifth nerve, which still persists in the case reported by Dr. Stiles.* If the respiratory centers are sufficiently affected, death follows from asphyxia. The difficulty in swallowing, combined with the frequently altered secretion in the throat, makes aspiration pneumonia a frequent cause of death.

Whether there are any disturbances of vision not due to the motor paralysis is uncertain. This matter is worthy of study when suitable cases afford the opportunity. No sig-

* Jour. Amer. Med. Assoc., December 27, 1913.

nificant retinal or optic nerve changes have been reported, but nearly all reports of epidemics mention various degrees of blindness, sometimes spoken of as amaurosis. Of course, the difficulty in fixation, the loss of accommodation, and the dilated pupils, not to mention the ptosis, are enough to make the patients complain of blindness. They do not sharply distinguish, as we do, between poor vision due to disturbances in the perceiving apparatus of the eye and difficulty in seeing due to the adjusting mechanism of the eye for focusing and fixation. As our patient said: "What difference does it make about my optic nerve? If I can't open my eyes or look at anything I am blind."

The mortality varies a good deal in different epidemics, as is natural from the character of the toxin and its variation under different conditions of growth, age, and possibly partial cooking. Heating to 180° F. destroys the toxin of botulism, unlike the toxins in other forms of bacterial meat poisoning. Thirty years ago 412 cases were collected by Senkpiehl, with a mortality of 40 per cent. Some epidemics show a mortality of 100 per cent., some as low as 10 per cent., or, rarely, no deaths; 50 per cent. is regarded as about the average today.

One of the most interesting things which came to light during the not very searching study I was able to give this case was the behavior of the ciliary muscle and its effect on the refraction. The following are the data on the refraction.

	O. D.			O. S.		
	Sph.	Cyl.	Ax.	Sph.	Cyl.	Ax.
Oct. 22,		+0.75	20°		+0.75	175°
Nov. 24,	−0.25	+0.75	20°	−0.50	+0.75	175°
Dec. 20,		+0.75	65°		−0.50	5°
Jan. 24,	−0.50	+1.50	85°		+0.62	90°
Mar. 15,	−0.25	+1.57	75°	−0.25	+0.87	95°
Apr. 14,	−1.00	+2.50	82°		−0.62	5°
Apr. 18,	−1.50	+2.75	80°	−0.25	−0.25	5°
			Under homatropin			
May 2,	+1.75	+0.25	90°	−0.50	+0.25	90°

The right eye, which required a cyl. of 0.75 in October to December, required a 1.50 cyl. in January and a 2.50 cyl. in April. The left eye, which required a 0.75 cyl. against the rule in October, required a 0.50 cyl. with the rule in December, equivalent to a change of 1.25 in the astigmatism.

We have to do here with a considerable change in astigmatism. A slight change might conceivably be caused by recovery of tone on the part of the external ocular muscles, including the difference in pressure of the upper lid on the cornea as the ptosis diminished. Examination with the ophthalmometer showed that there was only a normal corneal astigmatism of about 0.50 D. Hence the 2.50 and 2.75 found in the right eye in April must be lenticular. If this were so, it should appear when measured by the Souter ophthalmometer. Moreover, it was natural to surmise that the change was due to a change in the action of the ciliary muscle. As the nuclear cells recovered from the damage by the toxin, some cells would recover better than others, and so the parts of the ciliary muscle under their control recover tone better than the other parts; from this would result an unequal tension on the suspensory ligament and a consequent unequal curvature of the anterior surface of the lens. This also should show in the examination with the Souter ophthalmometer. Moreover, cycloplegia by homatropin should result in disappearance of the astigmatism if it were a so-called dynamic astigmatism, due to the unequal action of the ciliary muscle. To spare the patient, who was good enough to submit to various tests which were more for our benefit than hers, only one eye was examined and retinoscopy was omitted, as the results were conclusive without it. Dr. Souter was kind enough to make the examination with his ophthalmometer and demonstrate it to me. It was surprisingly easy and convincing. The anterior surface of the cornea, as already tested, showed only 0.50 astigmatism; the posterior surface of the lens was practically free from astigmatism; the an-

terior surface of the lens, which rarely shows an appreciable astigmatism, showed over 1.00 D. As homatropin and cocain had been instilled twenty-five minutes before the examination, the ciliary muscle had already begun to relax or a greater amount would have been found. Subjective tests, later on, when cycloplegia was practically complete, showed O.D. sph. $+1.75 \supset$ cyl. $+0.25$ ax. 90°. Here we have proof such as has never been offered before of unequal contraction of the ciliary muscle. But the conditions are excessively pathologic. Those who still advocate the theory of Dobrowolsky that unequal contraction of the ciliary muscle—dynamic astigmatism—is an every-day affair have only to bring forward a series of normal cases in which the phenomenon has been demonstrated by the methods here used.

BIBLIOGRAPHY.

For a survey of food poisoning, to place botulism in its proper perspective from that point of view, consult Bacterial Food Poisoning by Dieudonné, of Munich, translated by Bolduan, of New York; also articles by Novy in Osler and Macrae, Modern Medicine, vol. i, and in Ref. Handbook of Medical Sciences (1914, vol. iv, p. 420).

For a survey of ophthalmoplegia, to place botulism in its proper perspective from that point of view, consult Uhthoff in Graefe-Saemisch-Hess Handbuch, vol. xi, under Ophthalmoplegia, page 472, and under Poisoning, page 164.

For the history, bacteriology, symptomatology, pathology, immunity, etc., consult the monograph by van Ermengem, of Ghent, in Handb. d. path. Mikroörg., Kolle und Wassermann, vol. iv, p. 909; also Leuchs, same vol., p. 939. van Ermengem was the discoverer of B. botulinus, and this article is the best single article on the subject.

Wilbrand and Saenger give a brief discussion of botulism under Ptosis due to Poisons, vol. i, p., 278.

For the recent American work on the subject see Wilbur and Ophüls, Arch. for Int. Med., October, 1914, p. 589, and Dickson, Jour. Amer. Med. Assoc., 1915, vol. lxv, p. 492.

All the above articles have copious bibliographies.

ISOLATED PARALYSIS OF THE CERVICAL SYMPATHETIC ACCOMPANYING GOITER.

H. MAXWELL LANGDON, M.D.,
Philadelphia.

The sympathetic nervous system consists of a chain of ganglia on each side of the spinal column, of fibers rising from the spinal cord, leaving with the roots and passing into the ganglia, from which branches pass to the smooth muscles of the viscera, blood-vessels, glands, etc. Its uppermost portion is unique in not receiving white or efferent fibers from the spinal cord above the inferior cervical ganglion, and it is with this portion that this article deals.

The structures supplied by fibers from the cervical sympathetic have been shown to be:

1. The radiating or pupil dilator fibers of the iris. The branches concerned here arise from the first, second, and third dorsal nerves, pass to the superior cervical ganglion, from there to the plexus, around the internal carotid artery and the Gasserian ganglion, through the nasal branch of the ophthalmic nerve, becoming its long ciliary branches and perforating the sclera.

2. The involuntary muscles (Müller's muscles) of the orbit and eyelids. These muscles were first described by H. Müller in 1858, and consist of unstriped muscle layers across the sphenomaxillary fissure and orbital groove and of similar tissue in each lid, that of the upper arising from the under surface of the levator palpebræ and in the lower lid from the neighborhood of the inferior oblique; each is inserted near the bone of the tarsus.

3. The blood-vessels of the mouth and face. These fibers are mostly, if not all, vasoconstrictor.

4. Lacrimal, salivary, and sweat-glands.

In 1841 Budge and Waller arrived at the conclusion that the pupil-dilating fibers had their origin in the spinal cord, between the exits of the sixth cervical and fourth dorsal nerves. This conclusion has been confirmed by Dastre and Morat, who placed the center in the medulla, and denied by Salkowski and Knoll, who placed it in the anterior corpora quadrigemina. The weight of experimental, as well as clinical, evidence favors the existence of a spinal center, but this has not been absolutely proved.

From the above description of the parts supplied by this tract it is obvious that if it be irritated, widening of the palpebral fissure, extrusion of the eyeball, dilatation of the pupil, and some increase of the secretion of the salivary, lacrimal, and sweat-glands may be expected, the opposite being found if the lesion lessens the stimuli.

The centers and fibers are so connected with other tracts that a lesion affecting this pathway alone is quite unusual, and is either traumatic, following stabbing, shooting, or cutting injuries, or from local pressure, as by enlarged lymph-glands, aortic aneurysm, or goiter. Oppenheim, quoting Heiligenthal, says that "Goiters need not be very large to compress the sympathetic. It is most easily affected by flat goiters, which extend more or less far back." Heiligenthal also points out that the cervical vessels and other nerves can make way for the enlarged gland, while the sympathetic is firmly fixed. In a case described by Holz swelling of the thyroid first produced irritation and then paralysis of the sympathetic.

In addition to the above given signs of paralysis of the cervical sympathetic, loss of development or hemiatrophy of the same side of the face has been observed. Heiligenthal thinks that this symptom is due to atrophy of the fatty tissue. Hertel believes that atrophy of the orbital fat produces the enophthalmos seen in cervical sympathetic palsy and not

paralysis of Müller's muscles. This opinion is based on experimental work on young rabbits. When, however, it is recalled that toxic cases of hyperthyroidism with much loss of flesh are frequently the ones with marked exophthalmos, whereas if Hertel be correct, the opposite might be expected, it seems more likely that changes in Müller's muscles are responsible for the alterations in the position of the globe. Jonnesco, by his operative work, has proved that section of the cervical sympathetic has, for a time at least, the effect of lowering the tension of the eyeball, and this has been confirmed experimentally by many observers.

The two cases to be reported have certain things in common which may be summarized before going into a more detailed description of the local conditions. Both are from the clinic of Dr. John K. Mitchell, at the Orthopædic Hospital and Infirmary for Nervous Diseases; both are married women; physical examination of each was negative so far as heart and other viscera were concerned; negative blood and urinalyses; negative Wassermann and negative *x*-ray of neck; each had a small, hard, simple goiter. Both cases were shown at meetings of the Philadelphia Neurological Society:

CASE I.—Mrs. M. H., aged thirty-seven, reported first in September, 1915, complaining of sudden swellings in various parts of the body, lasting irregular intervals of time, not painful, and no cause being known. It was believed to be a manifestation of angioneurotic edema. The ptosis of the right eyelid was noticed, and the writer was asked to examine the ocular condition, with the following results: V. O. D. = 6/60, with a pinhole = 6/15; O. S. = 6/6.

The ocular rotations were full and equal in all directions, with no diplopia. The media were clear, discs a good color with clear margins, no fundus changes, with high astigmatism in the right eye. Fissure, R. E., 8 mm.; L. E., 9 mm.; pupil, R. E., 2 mm.; L. E., 3 mm.

On Hertel's instrument the right eye protruded 13 mm.,

the left eye 13½ mm. After instilling in each eye three drops of a 5 per cent. solution of cocain, the fissure of R. E. equaled 8 mm.; L. E., 10 mm.; pupil, R. E., 2 mm.; L. E., 5½ mm.; exophthalmos, R. E., 13 mm., L. E., 14 mm., making it seem without a doubt that there was a paresis of the right cervical sympathetic. This diagnosis was strengthened when the accompanying right facial hemiatrophy was considered. On questioning, the patient said this condition has been present about five years, and she believed it had recently increased, though this was not, as has been above stated, the condition for which she had sought advice.

With the Schiötz tonometer, after instilling a 2 per cent. solution of holocain, the tension in each eye was found to equal 22 mm. of mercury.

CASE II.—Mrs. E. G., aged forty-eight, gave a history of drooping of the right eyelid for thirteen years. There was no history of trauma. She thinks there has been little change recently. She had been told by an oculist that she had a partial paralysis of the third nerve. V. R. E. = 6/15; L. E. = 6/15. Ocular rotations were well performed; no diplopia with a red glass; media clear; discs a good color; no fundus changes; hyperopia, 1.5 D. Fissure, R. E., 4.5 mm.; L. E., 7 mm.; pupil, R. E., 1.5 mm.; L. E., 2.5 mm.; exophthalmos, R. E., 15.5 mm.; L. E., 16.5 mm.

After instilling three drops of a 5 per cent. solution of cocain and waiting thirty minutes the following changes were noted: Fissure, R. E., 5 mm.; L. E., 9 mm.; pupil, R. E., 1.5 mm.; L. E., 4.5 mm.; exophthalmos, 15.5 mm.; L. E., 17 mm. This, together with the right facial hemiatrophy, seemed to make positive the diagnosis of paresis of the right cervical sympathetic.

With Schiötz tonometer, after using a 2 per cent. solution of holocain, the tension in each eye was found to represent 20 mm. of mercury.

In view of the entire absence of other neurologic findings, it seems that the involvement of the cervical sympathetic in each of these cases is isolated, and, considering the negative findings in other structures, the accompanying goiters seem the most likely factors in the production of the palsy.

PARALYSIS OF DIVERGENCE.*

ELLICE M. ALGER, M.D.,
New York.

If we are to judge from the number of cases reported in contemporary medical literature, paralysis of divergence, or indeed any of the so-called fusion paralyses, must be extremely uncommon. Since my attention was first directed toward the subject, nine cases have fallen under my observation. I feel pretty confident, however, that many cases pass unrecognized, partly because we have not had the condition in mind as a clinical entity, and partly because the definite diagnosis of ocular paralyses requires more time and better facilities than are usually available in clinical work. The movements of the eyes result from impulses originating in or controlled by at least three different types of nerve-centers. First, there are the motor centers, impulses from which are conveyed to individual muscles through the cranial nerves. Presiding over these are the cortical centers, through which we have the power of turning our eyes voluntarily in various directions. Finally, we have the so-called coördination or fusion centers, which, acting through the motor centers, automatically adjust the position of the eyes with regard to each other so that none of our voluntary movements shall result in double vision.

Lesions involving the cranial nerves are common enough and are easily recognized. They cause a motor paralysis of the muscle or muscles of one eye supplied by the affected nerve. Lesions of the appropriate cortical areas, as in hemi-

* Candidate's thesis for membership accepted by the Committee on Theses.

plegia, result in inability to turn both eyes together to the right or left, or up or down, as the case may be, but they do not paralyze the muscles themselves, which are still capable of responding to stimulation from other sources, as in overcoming prisms.

The so-called fusion paralyses are entirely different. We know that in certain individuals the fusion *impulse* is congenitally absent or latent; that in others it is very weak, while in still others it is capable of being suppressed or even destroyed, but as to its anatomic seat we have not the remotest idea. The fusion *impulse* may have a single localization, but the fusion *performance* is probably divided between several centers not necessarily very close together. For instance, we have occasionally cases in which the convergence power of the eyes seems to be entirely wanting, not because of any weakness of the muscles themselves, for the eyes can be rotated in various directions in a perfectly normal manner, but because the power of combining the action of the two interni in convergence has been lost. There seems to be a distinct center for convergence, for it may be lacking in patients whose other fusion powers are preserved. There seems to be also a divergence center coördinating the action of the two externi in the act of recovering from convergence or in overcoming prisms. It seems probable, too, that there are several other centers whose existence is so far merely a matter of hypothesis, awaiting the clinical proof of convincing illustrative cases. For instance, a right hyperphoria, whether natural or caused by the action of a prism, results in a vertical diplopia, which is suppressed by the coördinated action of the right depressors and the left elevators. Indeed, there may be a double center coördinating the action of the right inferior rectus and the left inferior oblique when the eyes are directed to the right, and the right superior oblique and the left superior rectus when they are directed to the left. A left hyperphoria is controlled by an

entirely separate group of muscles, and may have a center or centers of its own, and the same may be argued of cyclophoria, the coördination of the inferior obliques in intorsion, and of the superior obliques in extorsion. Savage insists that there are at least twelve of these fusion centers. So far as I know there is no clinical confirmation of these centers by the definite paralysis of the coördination of muscles which are themselves not paralyzed at all, except in the case of convergence and divergence. The others may be much less common. They would certainly be much harder to prove conclusively beyond contravention.

Divergence paralysis implies that a patient who can use either externus in a perfectly normal way in rotating his eyes voluntarily to the right or left has suddenly lost the power of combining their action in recovering from convergence. That keen clinical observer, Parinaud, reported the first case in 1883;[2] the second case was also seen by him in 1889,[3] but the whole subject was given new life by the scholarly paper of Duane,[4] who pointed out the definite clinical symptoms which make it easy to recognize, and on the basis of which the symptoms of other fusion paralyses as yet unobserved can almost be foretold.

Diagnosis.—As in all ocular paralyses in which the diagnosis is to be beyond dispute, *the onset must be sudden.* The chief subjective symptom is *diplopia*, which, from the resulting disturbances of orientation and projection, is frequently attended for a time by vertigo and nausea. The diplopia is a characteristic one. The divergence power being lost, the visual lines of the eyes tend toward each other, and according to Sherrington's law relaxation of one function, like divergence, automatically results in the stimulation of the opposite function of convergence. At any rate there is a *convergent squint with homonymous diplopia* when looking at distant objects. As the object of fixation is brought closer to the eyes, the images keep getting closer and closer to-

gether until finally, at what Duane terms the "*point of equilibrium*," the visual lines cross and the double images are fused. If the object is carried still closer, the patient is able to maintain single vision by aid of his convergence power up to a point a few inches from the root of his nose. Within this point diplopia again occurs, because the limit of convergence power has been reached and this diplopia is "crossed." Duane has called attention to the fact that, when the object of fixation is approached to the eye till "*single vision by approximation*" is reached, the object can then be gradually withdrawn some little distance without diplopia recurring, and speaks of this as "*single vision by recession*." This was present in all my cases, and can perhaps be accounted for in this way: Paralyses of divergence automatically result in stimulation of convergence as the fixation point is approached to the eyes. When, however, it is carried away from them, convergence is automatically relaxed, and, though in this case it cannot be accompanied by any increase in the paralyzed function of divergence, the very relaxation permits some single vision by recession.

When the patient looks straight ahead, neither externus receives any stimulation owing to the paralysis, but if the object be carried to *either* side, the externus on that side begins to receive its normal stimulation from the cortical centers for voluntary rotation which are not involved, and *the diplopia perceptibly diminishes* and in many cases even disappears entirely. This is, of course, quite different from the diplopia in motor paralysis, in which the rotation of the eyes in the direction of the weak muscle causes a tremendous increase in the amount of diplopia.

Objective Symptoms.—When the patient looks at a distant object, he fixes it with the eye which has the best vision while its fellow *turns in* toward the nose, though not necessarily very markedly. If the fixing eye be covered, it turns it behind the screen, while the other can be seen to make an

outward movement of redress, as it in turn takes up the task of fixation. As the screen is shifted from one eye to the other it can be seen that this *deviation behind the screen is equal in the two,* while in motor paralysis the difference is very perceptible. As the object is brought closer to the eyes both the deviation behind the screen and the movement of redress are seen to be smaller and smaller till the position of equilibrium is reached, when they cease entirely. Within this point the cover test shows a reversed (inward) movement of redress, at first hardly perceptible, but becoming very pronounced as the object is carried to the base of the nose. The voluntary movements of the eyes to the right or left in divergence paralysis should be perfectly normal. When roughly tested, the patient can move either cornea clear to the external canthus, and the more delicate tests of rotation with the perimeter or tropometer show no muscular weakness of either externus.

Differential Diagnosis.—The conditions with which divergence paralysis is most likely to be confused, and their chief differences, are as follows:

Conjugate Cortical Paralysis.—Both eyes may deviate to one side, but there is no apparent squint and no necessary diplopia. The patient cannot rotate both eyes together much beyond the midline in one or more directions, but convergence and divergence are preserved.

Paralysis of one externus shows a greater diplopia, which increases tremendously as the fixation object is carried into the field of the paralyzed muscle and disappears entirely on the other side. The limitation of motion is marked by all tests, while the deviation under the cover test is much greater in the sound eye than in the diseased one, and increases as the object is carried to that side.

Paralysis of both externi is characterized by much greater squint and wider diplopia. The diplopia increases instead of diminishes as the object of fixation is carried to either

side, and there is inability to rotate either eye outward much beyond the midline.

Convergence excess generally occurs in young subjects, and the condition is not a stable one, varying widely from day to day. Both diplopia and squint are much less marked for distant than for near tests, and are much increased if the accommodation is brought into play. Rough tests show no change in the rotation of the eyes, but careful measurements in all but the most recent cases show increased inward rotation and decreased outward.

Divergence insufficiency is practically always the result of a previous convergence excess and has many of its symptoms. It is a fatigue condition, and is therefore neither sudden in its onset nor stable in its manifestation. The diplopia is less for distant objects and greater for near, and is very much increased by the use of the accommodation. It becomes less or disappears entirely after rest. Duane calls attention to the fact that the patient can maintain "single vision by recession" much further from the eyes than in paralysis of divergence.

Causation.—In most of my cases there was the probability of an organic brain lesion of some sort. The first probably had a small cerebral hemorrhage. He had a chronic nephritis with very high tension, and during the preceding summer had an attack of unconsciousness lasting several hours. Case 2 showed marked evidences of cerebral syphilis, while Case 3 was beyond doubt tabetic. Case 4 was diphtheritic, the first to be reported from this cause I think. Of Duane's personal cases, half were apparently due to organic brain disease, there being definite history of tabes, syphilis, multiple sclerosis, or brain tumor, while in the majority of the cases he collected from other sources the condition was so sudden and permanent as to justify the idea of a localized cerebral hemorrhage.

Treatment.—In ordinary motor paralysis the patient soon

learns to distinguish the true from the false image, and after a time learns either to suppress the latter entirely or to carry his head in such a position as to avoid the diplopia. At any rate, after a variable length of time all his subjective symptoms disappear, and it is often quite a task to make him conscious of his diplopia long enough for examination purposes. In divergence paralysis, however, the patient always has double images for distant objects which he can easily fuse at his reading distance, and it seems much more difficult for him to learn the psychic trick of suppression. Some of my patients either recovered entirely or learned it, but in others the diplopia persisted unchanged for years. In a number of Duane's cases the diplopia also persisted for long periods. It is, therefore, necessary to do something more than treat the cause of the condition. The most obvious resort seems to be the use of a prism base out before each eye. This plan of treatment is applicable only to cases where the diplopia is a moderate one, and such prisms should, of course, be used only for distant vision, since their use for near would tend to provoke the very convergence excess which it is our particular wish to avoid, as in my seventh case. If they are necessary at all for near work, they should be made as weak as possible. Another point of importance is the correction of the hyperphoria, which seems to be present in a surprising proportion of the cases. Most latent squints result from refractive errors, and are kept under control by the constant use of the fusion powers. It stands to reason then that, in divergence paralysis in which an important fusion element has been permanently destroyed, an unusually careful correction of the refraction should be made. It would seem that these cases, when of organic origin, were particularly suited for operative treatment after the laspe of a period of observation. The only operation that seems logical is the resection or advancement of both externi, attempting to secure even a divergence in such cases as have convergence

excess. Such a procedure would carry the patient's point of equilibrium off to infinity, while by using his convergence power he would be able to maintain single vision up to points close enough for ordinary near work.

CASE I.—Mr. X, aged fifty-six years, referred by Dr. D. G. Sheehan, consulted me in November, 1909. For a week he had been having a severe attack of facial neuralgia which his physician had partially relieved with phenacetin. During the last two days the pain had practically ceased, but he had become conscious of a very annoying diplopia which he proceeded to describe. A man a block away on the street appeared to him like two men some distance apart, but as they approached him they kept getting closer together and at last merged into one. He had no diplopia for near objects, and had been able to read his paper without difficulty. He also noted the fact that, by turning his head in either direction, he could for a time at least suppress the diplopia for distance. He had been having a good deal of vertigo and nausea, which are so common in disturbances of the ocular equilibrium, but which he ascribed to his neuralgic attack.

Examination of his eyes individually showed in each a moderately well-developed senile cataract. The fundus in each showed changes characteristic of arteriosclerosis: narrowing of arteries, indentation and tortuosity of veins, and one or two very small hemorrhages in each. His pupillary reactions were normal. His vision was somewhat reduced, being only 20/40 in each and not improved by glasses, though he was able to read fine type without much difficulty at the reading distance with his presbyopic correction.

Examination of his binocular vision showed a homonymous diplopia by all tests of 16°, the fixation light being 20 feet away. The diplopia was greatest in the primary position, diminishing and then disappearing entirely as he turned his head to the right or left. He had a right hyperphoria of 1.5°, which did not change materially as he moved his eyes. As the test light was approached to him the diplopia became less and less and vision became single at ten inches. If the light was carried closer to the eyes than this, the screen

test showed exophoria, while when the light was carried gradually further away he could maintain single vision some distance beyond the point where it occurred when the light was brought toward him. Measurement of the rotation of his eyes with the tropometer showed in the right an inward rotation of 50° and an outward one of 40°, and in the left an inward of 55° and outward of 45°, indicating a very moderate esophoria, but certainly no paralysis of either externus. These conditions were pretty constant during the week or ten days he was under my observation. As an actor with serious professional responsibilities, he shortly went with his company on the road, and I never had an opportunity to examine his eyes again. He wrote me, however, many weeks later, that the diplopia had ceased to annoy him, and that under treatment of his physician he was in apparently very good health. He died suddenly of pneumonia while on tour.

CASE II.—Seen in my clinical service at the Post-Graduate Hospital. Sarah G., a widow aged thirty-five years, born in Russia, had lost two children of diphtheria and her husband of tuberculosis. For two years she had had strabismus with diplopia, and had floated from one dispensary to another.

Examination of her eyes showed media clear and fundi normal and vision 20/30 in each. Argyll-Robertson pupils. She had homonymous diplopia, securing single vision at 20 feet only by aid of a 25° prism base out. The diplopia diminished as she turned her eyes either to the right or to the left, and also when the light was brought closer to them, vision becoming single at a point four inches in front of her. If at this point the light was gradually withdrawn, she could maintain single vision up to 15 inches. Cover tests showed, when the fixation point was closer than four inches, a divergence, while at all points beyond this there was convergence of the visual axes. She had a right hyperphoria of 3°. Her knee-jerks and other reflexes were greatly exaggerated, and though never able to get a Wassermann done, Dr. H. C. Cornwell was able to make a clinical diagnosis of cerebral syphilis before the patient disappeared from view. Neither was any exact measurement made of her powers of rotation, but clinically they appeared fully up to the normal.

CASE III was also seen in my service at the Post-Graduate Hospital. David S., aged forty-two years, had syphilis twenty years ago and had two years' treatment with inunctions and injections. Two years ago he began to have trouble in walking. Has lightning pains in various regions, an Argyll-Robertson pupil, static ataxia, and complete absence of patellar reflexes. A year ago he was suddenly conscious of double vision, with nausea and vertigo. An optician fitted him out with a pair of 4° prisms base out, and as he sees very much better with one eye than the other, the diplopia has not been very annoying.

Examination of his eyes showed him to be myopic in each eye, but there were no abnormalities in either vision or fundi beyond those due to the myopia. He showed a homonymous diplopia of 12° by all tests, with a right hyperphoria of 1°. The diplopia diminishes when the object of fixation is carried to the left or to the right, but does not disappear entirely. As a pencil is approached to him he sees it singly for the first time at a distance of 12 inches, and can maintain this single vision until it is withdrawn to 20 inches. Tipping his head forward lessens the diplopia. Objectively he shows a moderate convergent squint which diminishes under the cover test as the fixation point is brought nearer, until at a point of seven inches away the eyes are in a state of equilibrium. At a point four inches away the cover test shows an exophoria. Measurements of his rotatory power on a tropometer are rather below normal in all directions, but have a proper proportion and certainly show no evidence of any motor paralysis.

CASE IV.—Mildred M., aged four years, first seen May 25, 1914. Two months ago the child had a mild attack of diphtheria and since that time swallows badly, regurgitates fluids through the nose, and talks with difficulty. A month ago she complained to her mother of seeing double.

Examination of eyes shows media clear and fundi normal in both. Pupils react normally. Vision 20/40 each, increased to 20/20 by + 1 sph. Reads 20/20 and with sph. + 4 reads J. 1. Paralysis of accommodation. Diplopia of 20°, homonymous, diminishing to 0 at six inches. Single vision by recession to 25 inches. Cover tests show the same

measurements. Diplopia does not become either greater or perceptibly less when the fixation object is carried to right or left. There is no hyperphoria or vertical diplopia. The lateral rotations were normal. The child was never brought back for further examination, and I assume it went the same course as most diphtheritic paralyses.

CASE V.—Miss E. C., aged sixty-two years, seen November 22, 1914. For the last five days has suffered from diplopia which came on suddenly over night and is noted only for distant objects and does not interfere with reading. Patient has a low compound hyperopic astigmatism, but gets normal vision with either eye with correction and reads No. 1 type. Pupillary reactions are normal. Media are clear, but both fundi show angiosclerosis, having notched, tortuous veins, and in the right eye a few small spots of exudate and in the left a small retinal hemorrhage. Homonymous diplopia of 20° at 20 feet, increasing *very slightly* eyes right, and diminishing but not disappearing eyes left. Also there is a perceptible left diplopia which does not increase eyes up or down. Single vision is obtained at six inches and maintained in recession to twelve. Even at this distance the diplopia tends to increase slightly when the fixation object is carried to the right, while it diminishes to the left. The rotations of the eyes to the right and left were not reduced in any way. I never saw this patient again, but her sister informed me afterward that the diplopia lasted for about two months and then disappeared (or was suppressed).

CASE VI.—Mr. F. T., aged thirty-four years, patient of Dr. M. J. Echeverria, seen May 1, 1913. Doubtful history of specific infection several years ago, for which he has been thoroughly treated by several competent men, including a course at the Hot Springs. His Wassermann was negative a year ago. At that time his right eye began to turn in with the usual diplopia, and this has gradually increased, but for the last few months has remained stationary. Wears exclusion pad. Primary position shows a homonymous diplopia of 60° diminishing slightly, but distinctly both eyes right and eyes left. No hyperphoria. Single vision at two inches and by recession as far away as six inches. The

squint does not increase in accommodation. Pupillary and other reflexes are normal. Tropometer measurements are 50 in and 40 outward in each eye. The media are clear and the fundi normal. He gets normal vision under atropin with sph. + 1.50 ⊂ cyl. + 0.37 ax. 90 in each eye. The motility is unchanged under cycloplegia.

CASE VII.—Mr. L. H., aged thirty-seven years. Seen September 18, 1912. Has been in the army in the Philippines and had venereal warts, but gives no history indicative of syphilis. Wassermann not obtained. Is a chauffeur for a wealthy man, and seven months ago noticed that approaching cars were double and he was embarrassed in deciding which to dodge. Consulted a refracting optician who gave him a sph. − 0.75 with prism 2° base out in the right eye, and sph. + 0.75 with prism 5° base out in the left, which he has worn constantly. Sometimes he sees double with them at distance and he has constant headache. His media are clear and fundi normal. He has normal vision in each eye with sph. + 1 ⊂ cyl. + 0.50 ax. 90 in each eye, but vision is distinctly better with either eye alone than with both together. His pupillary reactions and reflexes are normal. He reads J. 1 with either eye. By all tests he shows a homonymous diplopia increasing slightly both to the right and the left. Diplopia diminishes as the fixation object is carried toward him, and disappears at six inches. Single vision by recession was noted but not measured. The ocular rotations were not reduced. I think in this case there was spasm of convergence resulting from wearing for several months prisms base in which were too strong for anything except driving his car. I gave him full correction of his refraction and an exclusion pad. I learned from a relative a long time afterward that his headaches ceased promptly, and that finally he ceased to be troubled with diplopia; but he never came back for reëxamination as advised, and it may have been merely suppressed.

CASE VIII.—Mrs. A. N., aged fifty-four years, referred by Dr. W. J. Carpenter. Seen December 10, 1914. Patient has been a widow for thirty years with several healthy children, and has had no illness of any sort except that left breast was removed two years ago for a non-malignant

neoplasm. Her urine is negative and her blood-pressure normal. Two months ago she began seeing double with some vertigo, but no headache or nausea. She has been myopic all her life, wearing sph. − 4.50 ⌓ cyl. − 0.50 ax. 180 and reading without glasses.

Examination showed media clear and fundi normal in both eyes, and her glasses were within a half diopter of her atropin correction, which gave her 20/20 in each. Pupils, accommodation and reflexes normal. By all tests she showed a homonymous diplopia of 9°, diminishing slightly both eyes right and left. Also a right hyperphoria of 2° not increasing. Single vision by approximation at 18 inches, by recession to 48 inches. Ocular rotations as measured by tropometer 45 inward and 40 outward for both eyes. She was put on K. I. on general principles, and the diplopia gradually improved. When last seen, on March 26, 1916, it had the same characteristics, but single vision by approximation was secured at 4 feet and by recession up to 10 feet, while at times when she was not tired she claimed single vision.

CASE IX.—Mrs. J. W., aged sixty-five years, consulted me on January 15, 1916, with the following history: A year ago she first noticed a persistent diplopia when looking at distant objects, but not present close at hand, as in reading. She is not in very robust health, but her physician, after thorough examinations, says there is nothing organically wrong. Examination of her eyes revealed the following condition: Has beginning senile changes in both lenses, but otherwise both eyes show normal pupillary reactions, tension, and normal media and fundi. With sph. + 2 she gets normal vision with each, and with presbyopic correction reads J. 1. By all tests she shows a homonymous diplopia of 8°, not increasing eyes right or left. Right hyperphoria 1°, not increasing eyes up or down. Single vision by approximation at 18 inches and by recession as far as 48 inches. The rotations of both eyes in all directions were entirely normal.

REFERENCES.

1. Barnes: Birmingham Med. Review, lxxxiii, 83, p. 232.
2. Parinaud: Archiv. Neurol., March, 1883.
3. Parinaud: Brain, iv, 1889.
4. Duane: Ophthalmology, October, 1905.

DIVERGENT CONCOMITANT STRABISMUS, THE MUSCULAR ANOMALIES AND REFRACTIVE ERRORS ACCOMPANYING IT, AND ITS TREATMENT.*

HERBERT WRIGHT WOOTTON, M.D.,
New York.

For several reasons an especial interest is associated with the study of the divergent form of concomitant strabismus. In the first place, as it is far less common than the convergent form, the etiology of its various types, in so far as relates to the muscular anomalies present, has been less thoroughly discussed. In the second place, the difficulties attending its rectification by surgical procedures are generally held to be greater than those connected with the correction of convergent strabismus. Lastly, amblyopia of the squinting eye defying improvement by correction of the refractive error is by no means as common, and, if we believe that the irremediable amblyopia of strabismus is for the most part congenital and not acquired, does not seem to present itself as an etiologic factor as frequently as in the convergent variety. While it must, of course, be admitted that a corneal opacity or a unilateral cataract, especially in an adult, will in many cases determine which eye shall diverge, it is certainly more common in divergent than in convergent strabismus, and particularly in divergent strabismus associated with bilateral myopia, to find the vision equal or nearly equal in the two eyes after correction of the refractive error.

Divergent strabismus is quite generally regarded simply as a condition diametrically opposite to convergent strabismus.

* Candidate's thesis for membership accepted by the Committee on Theses.

We are early taught to comprehend thoroughly, and subsequently we carefully remember, the etiology of the convergent type as elucidated by Donders, and based upon the disproportion existing between accommodation and convergence, but we are too apt to record the etiology of divergent strabismus as simply just the contrary. We forget that, while one muscular anomaly alone, a convergence excess, is causative in convergent strabismus, such is not the case in all instances of the divergent type. It is, I think, on account of a very general lack of recognition of this latter fact that divergent strabismus is regarded by many as so much more difficult to rectify than is convergent strabismus.

If we seek to classify cases of divergent strabismus according to their etiology, that is to say, according to their accompanying muscular anomaly, we find that they fall, as would naturally be expected, into three distinct groups. First, those accompanied by an insufficiency of convergence; second, those accompanied by a divergence excess, and, third, those presenting both these anomalies in varying proportions. If we attempt to base the classification upon the accompanying refractive error, we again divide the cases into three groups—those in which both eyes are myopic, those in which both eyes are hypermetropic, and those in which one eye is myopic and the other hypermetropic. The second classification is that usually adopted, and, from the viewpoint of etiology and treatment, it is exceedingly unsatisfactory. It takes no cognizance of the fact that any one of three muscular types may be present, usually assumes that all cases of divergent strabismus are accompanied and caused by a convergence insufficiency, and most frequently predicates one surgical procedure in all instances. On the other hand, a classification based solely upon the existing muscular error, while of much greater service as indicating the operative measures to be employed, is not entirely perfect, inasmuch as the different muscular errors are not invariably

associated with a constant error of refraction. Its practical value, however, as a guide to treatment cannot be overestimated, and the fact that, in the vast majority of cases, there *is* a constant relation between the muscular and refractive errors, constitutes, in my opinion, a strong plea for its adoption. As a very general rule, to which there are few exceptions, insufficiency of convergence is associated with bilateral myopia, divergence excess is associated with bilateral hypermetropia, and a combination of both errors in marked degree is usually found when one eye is hypermetropic and the other myopic. A lesser degree of convergence insufficiency or divergence excess may, of course, complicate the predominating muscular error in either hypermetropic or myopic divergent strabismus. On the basis of the predominating muscular error, we may therefore attempt the classification of divergent strabismus as follows. In the subjoined table both the muscular anomalies and the refractive errors are arranged in the order of frequency in which they occur, at least in New York city:

1. Divergence excess	Hypermetropia frequent. Myopia rare.
2. Convergence insufficiency	Myopia frequent. Hypermetropia rare.
3. Divergence excess (marked) and convergence insufficiency	Antimetropia frequent. Myopia and hypermetropia less frequent.

By regarding divergent strabismus from this viewpoint we obtain important indications in regard to operative measures, and can readily determine, as will be shown later, how much or how little benefit is apt to accrue from correction of the refractive error. Some surprise may be expressed that I have rated divergence excess and hypermetropia as the most frequent muscular anomaly and refractive error accompanying divergent strabismus, but such, I am con-

vinced from the study of a large number of cases, is the position they occupy in New York. In this city divergent strabismus associated with bilateral myopia and convergence insufficiency is comparatively rare. Cases of bilateral myopia, exhibiting convergence insufficiency as a latent error (exophoria), are fairly common, but it is only occasionally, and then for the most part when the difference in the refraction of the two eyes is great, that they develop into an actual deviation.

The determination of the muscular anomaly or anomalies existing in any case of divergent strabismus is abundantly simple. In uncomplicated cases of divergence excess the deviation exists only when the patient gazes at distant objects, or, if it be present, though to a less degree for near-points, the power of convergence can be shown to be unimpaired by insisting that the patient shall attempt fixation of some test object, such as the point of a lead-pencil, gradually brought nearer to him in the middle line. In typical cases, which are very frequent, he will maintain bilateral fixation until a point within three or four inches of the root of his nose is reached. Obviously, when this is the case, convergence insufficiency cannot be present. In uncomplicated convergence insufficiency the squint will be more marked for near than for distant objects, one eye diverging more and more as the pencil approaches the face. When both divergence excess and convergence insufficiency are present, the deviation will be marked during both distant and near vision, and the power of convergence will either be entirely absent or will be manifest only to a slight degree. In all cases of pure divergence excess the extent of the external rotation of each eye will be found to be markedly increased, and not infrequently comprises an arc of 70 degrees.

The nature of the refractive error associated with these muscular anomalies is so nearly constant that we are able with considerable certainty to hazard a guess at the char-

acter of the former from observation of the latter. For example, if the converging power is well preserved, if the deviation is present only when the patient gazes at distant objects, and if the power of external rotation is markedly increased, the refractive error is almost certainly bilateral hypermetropia. If, on the other hand, the converging power is markedly diminished or absent, and the squint is absent or slight for distant objects, bilateral myopia very generally coexists. Cases in which the deviation is marked for distant objects, and in which there is but little or no power of convergence, are very generally those in which one eye is myopic and the other hypermetropic, at least in one meridian; or if bilateral myopia should be present, the disproportion in the refractive error of the two eyes will be great. These rules have some exceptions, but they are few in number. I have encountered within the past two years two cases in which bilateral myopia existed and in which an almost pure divergence excess was the associated muscular anomaly. In both bilateral fixation was secured by a free tenotomy of the externi. It must also be admitted that convergence insufficiency may occasionally be the predominating muscular error when bilateral hypermetropia exists, although such an association is exceedingly rare. In the vast majority of cases the above table represents the relative frequency of the muscular and refractive errors according to my experience with divergent strabismus in New York city.

In the treatment of divergent strabismus it is generally held that our first endeavor should be to correct the refractive error and that, after such correction, a cure may result without resort to operative measures. A cure may thus be obtained, but only in those cases in which myopia fairly equal in degree is present in both eyes and in which convergence insufficiency is the sole, or greatly predominating, muscular anomaly. Correction of the refraction produces no effect when the deviation is due to a divergence excess.

It follows, then, that most cases of divergent strabismus necessitate operative measures for their cure, and the results are, generally speaking, exceedingly satisfactory and frequently perfect if we are guided in our choice of operation by the muscular anomaly present. The surgeon who makes it his practice to employ in all cases the same operative procedure—advancement of the interni, tenotomy of the externi, or advancement of the internus and tenotomy of the externus of the squinting eye—is certain to meet with a greater number of failures than he who varies his method to accord with the muscular error he is called upon to attack. The cases most frequently encountered in New York are those of bilateral hypermetropia associated with divergence excess. In these, advancement of both interni will result in failure. The proper procedure consists in a *free* tenotomy of both externi, repeated, if necessary, and this is no less true when, as is rarely the case, bilateral myopia is the refractive error. In the hypermetropic cases, even if a fairly well-marked convergence insufficiency coexists, the tenotomies should constitute the primary measure and should be persisted in until the deviation is corrected for distant vision. When bilateral fixation for distant objects has been thus obtained, it will not infrequently be found that the power of convergence has returned to normal and that further interference will be unnecessary. Cases of myopia associated with convergence insufficiency, which persist after full correction of the refractive error, require the advancement of one or more generally of both interni, and in no circumstances should tenotomy of the externi be performed in pure cases of this type. When both divergence excess and convergence insufficiency in marked degree are associated, the predominating muscular error, which, except in cases of bilateral myopia, is usually the divergence excess, should be first attacked by means of free tenotomies of the externi.

COLOR INTERLACING AND PERIMETRY.*

CLIFFORD B. WALKER, M.D.,
Boston, Mass.

In all subjective examinations the factors of personal equation, either on the part of the patient or on the part of the examiner, are often very difficult, sometimes impossible, to control sufficiently to obtain absolutely reliable results. Particularly is this true in the examination for color interlacing, where strong personal equation must be contended with not only on the part of the patient, but also on the part of the observer. Although there are various other factors which may contribute variation in the result, the double entry of personal equations doubtless adds the major part of the unknown in the result.

On the part of the examiner the personal equation is largely the result of the method of examination, and his analysis of the mental and physical condition of the patient and also of the mechanics and physics of perimetry. There is also involved a certain judgment of the accuracy of the patient's answers, acquired only after considerable experience.

On the part of the patient the personal equation is largely the result of the mental and physical condition. In both patient and examiner previous experience is an important factor.

It becomes necessary, then, in speaking of the finer perimetric changes, to outline details of the method used and of mental and physical analysis on which it is based. This discussion may be carried on more or less in the order in which

* Candidate's thesis for membership, accepted by the Committee on Theses.

the various factors may present during the course of an examination. In this way the factors to be considered may be roughly grouped in order under the following heads:

1. Psychologic.
2. Physical or physiologic.
3. Mechanical.

Psychologic.—Considering a person of whom the examiner has no previous knowledge entering the "vision room," the first step is to get an idea of the person's mental capacity, while the visual acuity, with correction if necessary, is being carefully taken and recorded on the chart as an important part of the field record. The acuity examination in itself may throw considerable light on the person's intelligence or may even disclose an aphasia. It always gives a clue as to what fixation ability may be expected and frequently will indicate the presence of a scotoma. A patient may read a line better backward than forward, and vice versa, thus giving a clue as to which side of the macular region is damaged most. This clue may be emphasized in the manner in which the head is tilted or the eye is deviated in reading.

The age, education, and occupation of the patient, in case these things are not already known, are brought out in the course of a few questions to get an idea of the intelligence of the patient.

It is only rarely that children not yet old enough to go to school can be examined perimetrically. Highly educated people, scientists, engineers, etc., are apt to be so overanxious to give the best possible readings that they may get to seeing things that are not there, as it were, and especially on colors these people, as well as artists and people familiar with colors, are able unconsciously to take advantage of the percentage of chance and also to judge colors entirely by their light intensity. This latter possibility comes about from the fact that the colored test-objects show a difference in brightness or intensity of reflection before they give a sensation of their

true color as they approach from the periphery. And more than this, they pass through a series of more or less neutral tints as they reach their threshold of true color perception. Most patients are conscious of this phenomenon, although their description of it will show individual variation. Thus, a blue spot when it is first detected as moving may give the same appearance as a white spot at the periphery for form; it then becomes a slate color, and turns rapidly blue as it comes farther in. Red, while it has usually a sharper threshold than blue, approaches through a similar series of neutral tints beginning with a gray, which becomes brighter and yellowish, even brown, before it rather abruptly turns red. Green has the most unreliable threshold apparently, passes gradually through gray, straw color, yellowish, and then rather slowly and uncertainly, as compared with the other colors, turns green as it approaches the center. For this reason green is not used so much on account of its unreliability as to reduce the percentage of guess to at least 33⅓ per cent., for most patients will begin to anticipate the next probable color when anything like a definite sequence or order of appearance is allowed to occur. This difficulty is readily overcome by an instrument which will be described briefly later. At best, however, the first set of readings is often wrong as compared with the later readings, because the patient has not standardized his judgments, and even when his judgments have become as constant as they will, it is necessary to take the average of a number of readings, since a single reading may vary considerably from the average. Indeed, when these results are plotted on the chart they probably have an appearance of greater accuracy and definiteness than is actually present.

For the best results the form field should be made a separate examination from the color test, because it involves a different class of judgment, but, of course, all the color readings on a particular radius should be taken as nearly at the

same time as possible in order to get the best possible judgments. The first few readings in both cases are apt to be wrong and must be discarded. Later averages must be recorded throughout. The reading for "white" is practically worthless because of its very confusing threshold, varying through all the shades of gray. For this reason the patient is never allowed to say "white," but is forced to report first motion only.

The instruction given a patient is very important. If measurements are attempted before the patient thoroughly understands the requirements, they are, as a rule, worthless. Instructions are tempered according to the mental capacity of the patient, and must be quite labored with the mentally deficient, obtuse, drowsy, or aphasic. Any previous experience the patient may have had is taken advantage of or corrected as necessary. Three ideas must be grasped by the patient, namely, to fix the center and name the color, or report the first motion of the discs, and it is usually an advantage to inculcate the latter two ideas separately as required. Generally the form fields are taken first.

The limits of fixation ability are first to be gaged by the visual acuity and by the mental status. With vision of 20/40 to 20/200, good fixation can be obtained by enlarging the fixation spot somewhat. If it is much worse than 20/200, special mechanical devices, later to be mentioned, may be necessary. With corrected vision worse than 20/40 some sort of central scotoma can usually be demonstrated with some of the smaller discs of the series (Fig. 1).

The rapidity with which the discs are moved has a considerable effect on the reading, depending on the reaction time and observation power of the patient. With patients who are sluggish and disinterested, the measurements may readily be too small if the movement of the disc is not rather slow and some oscillatory motion is given to the disc while the attention is continually stimulated. A great many

more acute individuals are, on the other hand, always straining for the best possible reading, and complaining that the disc moves a considerable distance before they can speak. This is usually found by repetition to be due largely by a psychologic impression and seldom represents much error. With many patients variations of 10 degrees to 15 degrees are easily gotten by changes in the rate of motion of the discs, especially with colors. The only way to control such variables is to find, by repeated trials, the speed at which the response is most satisfactory, and use that speed con-

Author's Series of Discs			Bjerrum Series		Chart Letters of Nearest Equivalent Visual Angle
Diameters in mm.	Visual Angles on 28.6 cm. Perimeter		Equivalant Values in Minutes	Ratio of Diameter Distance in mm.	
	In Degrees	In Minutes			
0.15	1/32°	1.8′	1.7′	1/2000	20/5
0.31	1/16°	3.7′	3.4′	2/2000	20/10
0.62	1/8°	7.5′	8.6′	5/2000	20/30
1.25	1/4°	15.0′	17.2′	10/2000	20/50
2.5	1/2°	30.0′	34.4′	20/2000	20/100
5.0	1°	60.0′	57.3′	5/300	17/200
10.0	2°	120.0′	114.6′	10/300	8/200
20.0	4°	240.0′	229.2′	20/300	4/200
40.0	8°	480.0′	458.4′	40/300	2/200

Fig. 1.—Disc series and equivalents.

stantly throughout. With considerable experience this is not difficult, but beginners are apt to have their attention distracted with other details and use marked variations in speed. It must also be borne in mind that patients, while looking at the center, have their attention focused along a certain arc along which readings have been or seem about to be taken. If now an object is moved quietly along an arc in the opposite part of the field, the patient will often ignore it for some time and give a very delayed reading. It is always fair to allow the patient foreknowledge of which arc

is to be used. Scraping or other noises associated with the motion of a test-object will frequently influence the reading, generally making them too large by straining the attention and stimulating psychologic impression unduly.

Unconsciously an examiner may more or less make the readings fit whatever preconceived idea of the field he may have. I have felt the subtle pressure of this sort of thing, and have accordingly sought to take the field before making any ophthalmoscopic examination or acquiring much information about the patient other than that a field was desired. Fortunately, I have been ideally situated, with plenty of time at my disposal to carry out such a procedure. The field examination has been almost invariably the first examination I have made upon a patient, the sole idea being to get an accurate field record quite independent of the findings of others.

Physical.—Probably the most important factor contributing to accurate observation on the part of the patient is physical comfort. Unfortunately, with brain cases, where perimetric examinations may be of most value, a comfortable position for any length of time during the examination is very difficult, sometimes impossible, to obtain. The patient should be seated in a comfortable chair, at an adjustable perimetric table, on which the patient may rest the arms, in case a suspension apparatus, such as herein described, is not available. A cold iron chin-rest seems to be a great annoyance to some patients, especially hypophyseal cases with subnormal temperature. We have therefore used a wooden chin-rest. The Helmholtz bit has been found too distracting and uncomfortable, not to say distasteful, to the patient. But still more objectionable is the fact that they cannot call the colors readily with the mouth so occupied, and pointing methods involve too much reaction time.

When fatigue comes on, as it is pretty sure to, before the examination is complete, in any of the more difficult cases

there is no alternative but to allow rest periods—in some cases quite frequently. Asthenia and dizziness may require the assistance of a nurse, with pillows and props in addition to frequent rest periods.

The presence of severe headache or pain always disturbs the concentration and attention of the patient, and is often very difficult to contend with. These factors give greater variation to the readings, so that the average must be taken between greater numerical differences. Accordingly, single sets of readings are much more apt to show the condition of color interlacing. Unfortunately, this group of cases are often the ones that have choked discs and other evidence of intracranial pressure, and must be decompressed at once, so that it is difficult to wait for the infrequent periods when the headache is very slight. The field then must be taken with frequent rest periods, since the attention becomes distracted by the pain. Those cases which have temporary amaurosis often have their rest periods well regulated automatically by the amaurosis. If the patient's energy must be conserved, even at a slight sacrifice, the form field may be taken at the same time as the color fields, that is, eight readings at a setting of the plain perimeter, or many more on the perimeter I have devised, later to be described.

Mechanical.—Not infrequently the room in which the perimeter is placed is quite unsuitable for the best work. Thus, it may be necessary to set up a perimeter in a noisy clinic or ward, or in a room where people are passing through. Any such place where the attention of both observer and patient is continually being distracted is extremely unsuitable, yet frequently found in use because of convenience, necessity, or because the examination is not considered worthy of special arrangements. In our work we have selected a room with large windows of northern exposure. The walls facing these windows are darkened, and the examiner wears dark or black clothes and gloves. Fields are

taken as much as possible during certain hours of the day, when the lighting is best, avoiding cloudy or dull days. We have almost always been able to get our work done during these hours, but have recently installed an indirect lighting system to virtually lengthen these hours by assisting the weakening daylight as occasion may demand (*cf.* Fig. 7). The temperature of the room must not be too hot or too cold, lest the patient's thoughts wander to physical discomforts.

To explain the form in which our perimetric instruments have been devised and used it will be necessary to outline what we believe to be the requirements for accurate perimetric measurements. First, with regard to the form fields. The discs should be rimless and wire handled, so that the motion detected by the patient is due entirely to the test-object and not confused with that of a carrier. The hand should be held well out of the field as the disc is made to approach the field of vision, perpendicular to the periphery at all points. Thus, if a quadrantal defect is encountered, it is necessary to test its margins at frequent intervals along perpendiculars to its lines of cleavage. It is not sufficient to get a reading of 80 degrees on the horizontal line and another at 10 degrees on the next radius, and simply connect those two points with a line. That region from 10 degrees to 80 degrees must be tested at frequent intervals in the same manner as the rest of the field. It often makes a considerable difference in the reading whether a disc is approached at right angles or tangentionally to a line of cleavage. Likewise in outlining scotomata the disc should be moved from the blind to the seeing field perpendicular to the visual margin. This may be quite difficult to do on the ordinary perimeter, but is very easily accomplished on our perimeter (Fig. 2). Again, in testing the color fields, especially with regard to the presence of color interlacing, it is necessary to be able to change the colors instantaneously without any noise of the instrument or associated motion of

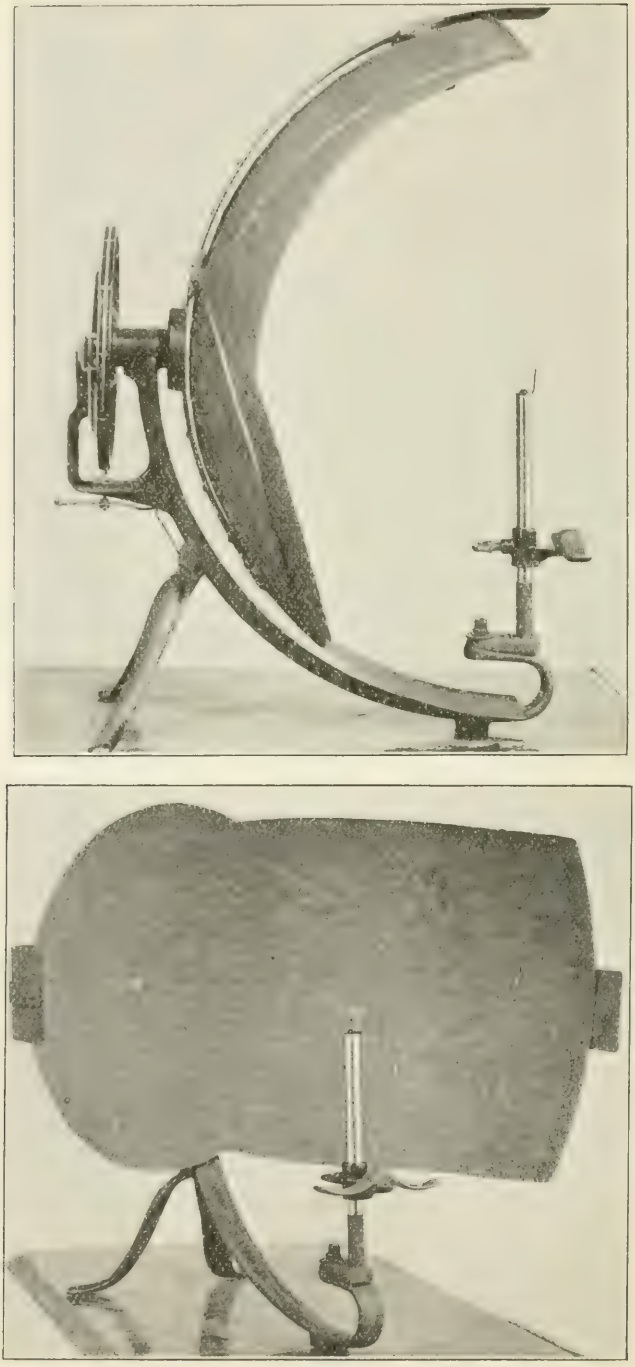


Fig. 2.—Special perimeter with wide movable face.

the hand by which the patient may anticipate or guess what has happened. To further reduce the chance to guess, at least three colors should be used, and they should be used in rather rapid succession, but seldom in the same order of sequence, the whole object being to obtain readings which represent as nearly as possible pure observation of color. We have previously described* an instrument to fulfil these requirements.

This instrument (Fig. 3) consists of fixed and movable parts attached to rods and finger rings in the general manner of a tonsillotome. The movable rod carries at the end an essentially rimless white disc, and farther back the colors, which abut each other to get quickest possible interchanging, are protected by slight lateral rims. On the handle end of the rod are notches which lightly engage a tension spring each time a color appears at the central display opening. The spring may be regulated in tension by a double set-screw, so the click on engaging a notch is not heard at all, but barely felt by the operator. At the two end stops for colors no click, of course, is necessary. To make the white spot appear requires more force than to display the colors, since in so doing more tension is put on the spring by means of an elevating cam. The ring on the handle for the thumb is on a swivel, so that any part of the perimeter is readily reached if the fingers are entered on the same side as the colors appear. In getting the form field the white spot is made to precede

Fig. 3.—Disc carrier and color interchanger.

* Walker: "Some New Instruments for Measuring Visual Field Defects," Archives of Ophthalmology, vol. xlii, No. 6, 1913.

the rest of the instrument, so that the first motion noted is surely not due to the handle. The colors ride very close to the beveled opening, so that practically no shadow is cast to cut down the area of the disc, and a slight allowance is made in the size of the opening to offset the light shadow present. The discs, protected as they are, do not fade or get soiled for months, and when necessary, they can be replaced by loosening a small thumb-screw on the handle, which allows all parts to be separated when the protective back at the end is slipped off. This instrument, called the color interchanger, in addition to serving well in rapid routine examinations, has also been used in testing for color interlacing, as will later be described.*

Fig. 4.—Practically rimless graduated discs.

The normal size of disc was always used in this instrument, but a separate series of discs for various purposes was gradually developed. Briefly, this series ranges in size above and below the normal or 5 mm. disc, so that any disc has one-half the diameter of the next larger and twice the diameter of the next smaller. Nine sizes have been found sufficient for a complete examination, the smallest being 0.15 mm. and the largest 40 mm. in diameter. These discs are essentially rimless, and two discs are carried at each end of a wire handle (Fig. 4).

* When this instrument is used on the new perimeter, it should be bent to the perimeter curvature or a little less.

Red and blue are back to back on one end, with green and white likewise at the other end. Color discs do not extend below 1.2 mm. in size. These color discs may also be used in testing color interlacing, as will be noted later. The white series above normal are used to detect the presence of what we have called "*response to large discs,*" as an index to state of optic block or atrophy in the areas blind to the normal disc. The smallest sizes are used to detect early changes within the field to the normal disc. A more detailed discussion of these phases may be found in a previous paper.*

Central scotoma cases usually develop a seeking motion of the eye in fixation. This motion, while it may be as rapid as a very slow nystagmus, is more commonly a sluggish, roving motion, with a tendency to come to rest in a slightly eccentric position for a variable length of time. If this motion is not controlled in some way, it is, of course, conducive to color interlacing readings. Although the preliminary mapping out of the blind spot, as a check on the fixation and observation, is a routine with all cases, it is an especially valuable procedure in central scotoma cases. If the scotoma is relative, or if the central vision is better than about 20/100, one usually enlarges the fixation spot somewhat and puts a good clear letter or number on it so that satisfactory fixation, as checked by the corneal reflex and the blind spot, may be obtained. If the central vision is about 20/200 or worse, one may obtain a satisfactory check on the fixation by use of our combination blinder and macular selector (Fig. 5), described in the previously mentioned papers. With this instrument the better central vision and fixation ability of the other eye are used to coördinately assist the eye with poor vision. If, however, both eyes have bad central scotomata, so that enlarged fixation discs will not

* Walker: "A Contribution to the Study of Bitemporal Hemianopsia," Archives of Ophthalmology, July, 1915, xliv, No. 4, p. 369. Transactions of the Ophthal. Section of the Amer. Med. Assoc., June, 1915.

give satisfactory results, even when assisted by tactile sense, one can still test the color-fields as to their relative arrangement, although their absolute arrangement with relation to the field and fixation point may have a constant error. One

Fig. 5.—Combination blinder and macula selector.

may arrange strips radiating from the center of the perimeter, and with the aid of tactile sense get the patient to fix on the point where these strips would seem to meet. Again, several spots may be stuck on the perimeter at points found by several approximations to lie just on the periphery of the

scotoma. The patient is then required to keep all these spots in view while the peripheral field is being examined.

As we have already mentioned, we prefer a plain perimeter, especially one of our own design, shown in Fig. 2. In stating this preference it is, of course, necessary to give our objections to the mechanical perimeters. First, as regards the color-fields, with practically all mechanical perimeters only one color-field can be taken at a time satisfactorily. This is quite favorable to the production of color interlacing, since the patient is not forced to distinguish the color, as must be done when several colors are used. In the Hare perimeter the colors can be interchanged mechanically, but the change is announced by a loud click, so that the patient has but to remember the simple sequence, and the conditions are similar, though better, than in the ordinary mechanical perimeter. Complete or a variable amount of foreknowledge concerning the color, as well as movements and sounds associated with the change of color, influences toward color interlacing readings. As has been stated in previous papers, the colors should be chosen to match the spectral tints.

Foreknowledge in taking the form field has to be contended with, as in the color-fields, but in a different manner. Some patients, in trying to detect the first motion of the disc in the region where they know it must approach, occasionally seem at times to mentally visualize the motion before it is actually seen. A psychologic error of this sort may be stimulated by a slight scraping of the test-object on the perimeter, or other associated noises or movements at this critical moment. This is a difficulty with most mechanical perimeters. Errors of this sort may be avoided on our perimeter by care in moving the discs, by having a large field to work in, by rapid repetition, and are made particularly noticeable when different sizes of discs are used in rapid succession, since the relation of their readings will often give an index as to which is decidedly wrong.

This naturally suggests further difficulties with the mechanical perimeters. In the first place, there is a marked tendency to record a particular reading, either the first or the last, either one of which may be wrong, rather than to grind back and forth and finally record a true average of the best readings. But, still worse, it is quite impractical to use the numerous different-sized discs in rapid succession; also rimless white spots cannot be used as we think necessary in modern perimetry. A mechanical perimeter, with which this could be done and move the discs in the manner previously mentioned, always perpendicular to the line of field clearage, seems quite impossible at the present time. Further, regarding the movements of the discs it must be noted that mechanical perimeters cannot move the disc past the center. In working out central scotomata, with or without a connecting field defect, it is necessary for rapid and accurate work to be able to move the disc past the center until it is seen or noted to grow brighter according to the character of the scotoma. Indeed, most mechanical perimeters are not at all reliable for measurements about the center, nor can they make measurements of more than 90 degrees, although the temporal field may be over that amount.

Electric perimetry has been recently advocated, but from our standpoint is open to severe criticism. Since it is but little used, only a few of the major difficulties need be mentioned. The worst and practically insurmountable difficulty arises from the fact that electric perimetry, to have any advantage over ordinary perimetry, must be done in the dark with a mechanical perimeter after dark adaptation of the retina has taken place. This means fixation in the dark, and I have yet to see the patient, especially a brain case, whom I could trust to look at the center throughout a complete field examination. The eye of the patient must at all times be observed by the examiner, otherwise gross errors will certainly occur. At least such has been the case in my

experience, so that for this reason, if for no other, I have discarded the method. But in addition to this major difficulty this method is heir to all the difficulties already mentioned in connection with mechanical perimeters. As for making a readable record with nine or even less different sized rimless discs and three colors by simply punching holes in the chart with any mechanical perimetric device, it seems simply out of the question. Even if these factors could be controlled, the phenomenon of dark adaptation is a troublesome factor. Although dark adaptation is largely accomplished in ten or fifteen minutes, it is not complete for from thirty to forty-five minutes. Any considerable flash of light, accidental or otherwise, during the course of the hour or two necessary to take a complete field means a further sacrifice of time for readaptation. Throughout, the sensitiveness for one size of disc will be different from that of another size, and also varies with the distance from the center. After-images also bother in this sensitive state, particularly from the fixation light or from a test light not in constant motion. It is hardly worth while here to take up, in addition, other difficulties, such as weakening batteries and lamps, pupillary reactions, drooping lids, or malpositions of the eye or head at the center post, dispersion and diffusion of the light in the vitreous, etc. Altogether, we consider electric perimetry far less accurate and far more time-consuming than daylight perimetry. It may be added, however, that a battery of electric lights may be satisfactorily used to augment waning daylight or even to take fields at night. We are arranging to use nitrogen lamps covered with milk glass and fixed in a circle back of the patient's head. A rheostated battery of this sort may be made to imitate daylight conditions quite closely for emergency work (Fig.6) .

For the practical examination of difficult fields we have devised a perimeter with the largest possible surface upon which to make measurements. This was accomplished by form-

ing a sheet of thin brass into a section of a sphere of 28.6 cm. radius. This surface was cut to extend 35 degrees about the center in three quadrants, and 95 degrees in the remaining quadrant, with a lateral width sufficient to include three of the 30 degree lines radiating from the center. The whole surface was marked by deep numbered lines, scratched in the metal to correspond with the field chart. As may be seen in the cut (Fig. 2), this section of sphere is attached to the ordinary perimeter by means of rollers, so that "eccentric fixation" may be obtained by displacement along the arc to the desired number of degrees. This feature is useful in obtaining the maximum periphery in spite of the obstruction offered by nose, eyebrows, lids, or cheeks. The position of the arc is set at any desired 30 degree angle, and fixed in position by a spring catch. The eye is placed at 28.6 cm. from the center in order that the discs shown in Fig. 1 may be recorded in visual angles directly as indicated or in diameters. The face of this perimeter is large enough, so that by use of the smaller discs the field for the smaller visual angles can be made with much greater ease than by the comparatively awkward and laborious method of Bjerrum. These specially made small discs and their use on the perimeter are fully described in the paper previously mentioned. With a series of discs and a perimeter of this design we have been able to completely work out difficult fields according to our requirements outlined in the shortest possible time.

With this rather brief but necessary sketch of our perimetric methods we may proceed to a study of the phenomena of color interlacing. As has been said, we began to take fields with practically no knowledge of the case. This was largely the result of the press of routine affairs. It became convenient to take the fields first, even before an ophthalmoscopic examination. The fields had, therefore, to be very carefully taken in order to get, if possible, a field that would stand checking and rechecking if later argument arose.

Fig. 6.—Special perimeter (of Fig. 2) mounted on suspension apparatus and artificially illuminated.

Before long we began using a wooden model of the described color interchanger until the instrument could be made in metal. While in one or two of our earlier fields we had found points of color interlacing, we were very rarely able to find interlacing after we began to use the color interchanger to get rapidly averaged readings. Indeed, in the few cases where it was found, papilledema or choked disc was commonly not present to account for the condition, but rather such conditions as central scotomata and poor fixation.

Accordingly, we began to study all the previous fields, from the very first, taken in the neurologic surgical clinic of Professor Harvey Cushing. A record was made in each case of the diagnosis, presence of papilledema, or choked disc, and its measurement in diopters, the presence or absence of color interlacing, operations, the mental condition of the patient, as well as the presence of pain, and various conditions which would have a bearing on perimetry. Also the name of the observer was recorded with each field as an index of whatever personal equation might exist on that score. Bearing on the question of color interlacing, 405 fields were found taken on 132 cases by 14 observers in the Johns Hopkins Hospital clinic, and 245 fields taken on 83 cases by 12 observers in the Peter Bent Brigham Hospital clinic.

Before proceeding to a discussion of the results of this study, mention should be made of the methods used by the various observers. The color tints used by all the observers were practically the same, being colored mat papers selected to match the spectral colors. Beyond this, the technic and equipment varied. The following procedure had long been in vogue at the Baltimore clinic. Green was rarely used. Red and blue circular discs, 5 mm. in diameter, were fixed on opposite sides of a black disc, about the size of a silver half-dollar. A standard plain perimeter was used. When one color reading had been made, the disc was either with-

drawn from the color field and rotated for the other color reading, or, where more care was used, it was withdrawn back of the perimeter arc and carried out to 90 degrees still behind the arc, to be again advanced on the face of the arc for a repetition of the previous reading or, if it had been rotated meanwhile, for a reading of the other color. The latter process occupied a longer time interval than was best, but was better than rotating the large disc while still readily noticeable to the patient in the peripheral field.

In our own, and largely among the observations at the Boston clinic, a variety of tests have been used in a routine manner. First, with the color interchanger, certain critical tests may be made. One may carry the red color to the periphery for red, and, while still holding the attention of the patient, noiselessly and quickly change to blue, when if inversion is present, no color will be called, otherwise blue will be at once detected. Green may be introduced in the same manner not only to find its periphery, but also to reduce the chance of guess. Again, the blue periphery may be found, then, when red is suddenly interchanged with the blue, it will not be called until it is brought nearer the center if interlacing is not present. Also the colors may be continuously and rapidly interchanged as the instrument is carried slowly along the arc, noting which color is first called. A very similar test may be performed with plain rimless discs. Red and blue or red, blue, or green discs may be fixed on the end of a rod, so that they can be made to approach the center abreast, and it is noted which color is detected first. In this case the colors must be detachable, so that the order can be rearranged each time. A series of *different sized colors* also offer a check, for it has been noted that, when considerable uncertainty is present with colors of 5 mm. and smaller, so that the readings are close together, with somewhat larger sizes the relations may become more distinct.

The main object of our tests is to force the patient to make color readings, which at all times require the color to be distinguished and differentiated, as a means of detecting and discarding the numerous errors that certain patients may make when wrought up by examination to the psychologic condition in which every moving object in the periphery apparently has some color, and also to detect in time the point where weariness or ennui drives the patient to random guesses and hurried answers in order to shorten the examination. But whenever readings are recorded at any time, they must represent the averages rather than single observations.

TABULATED REVIEW OF FIELDS FOUND IN JOHNS HOPKINS HOSPITAL RECORDS.

It was found very difficult to tabulate and present in a reasonable extent of space the large amount of data gathered in connection with the study of the great number of fields taken in Professor Cushing's Baltimore Clinic, and having relation to the subject of color interlacing. After struggling with this data for a considerable period it was decided that the following arrangement would perhaps give a satisfactory amount of information. In the first column the surgical number of the first admission is given, and readmissions are noted in the second column, which is used for the most part to indicate whether the field was taken before or after operation. All cases were recorded as "before" until an operation was performed, and then recorded as "after." Sometimes no operation was performed and sometimes no fields were gotten after operation, for various reasons, so that the number of "befores" considerably exceed the "afters." The operation, if performed, was always some form of decompression—transsphenoid, subtemporal, or suboccipital. In the third column is placed the diagnosis. In the fourth column is placed the condition of the optic disc, using the abbreviations "C. D." for "choked disc," and "O. U." for

"both eyes." Likewise, in the fifth column the presence of color interlacing in the fields is indicated by the letters "C. I.," and its absence by "No C. I." If two fields are taken by the same observer, and showing the same characteristics, it is noted in the sixth column as "2 f." In the last column the examiner who took the field is indicated by an arbitrary letter chosen at random.

To segregate the cases into well-defined groups was also found difficult, because of the numerous cases which would appear in more than one group if the differentiation was at all strict. Accordingly, to avoid placing a case in more than one group it was classified with respect to the predominant characteristics under discussion, and the complete record was placed under one number—the surgical number of the first admission. This arrangement seemed to render variations more easily traceable than to record the same case in more than one group. Apparently the following five groups were as satisfactory as any that could be arranged:

I. Optic discs normal or with atrophic pallor, but often showing color interlacing.

II. Optic discs edematous, though not measurably elevated, but often showing no color interlacing.

III. Optic discs edematous, though not measurably elevated, but often showing color interlacing.

IV. Optic discs measurably elevated without color interlacing.

V. Optic discs not measurably elevated with color interlacing present in one or both eyes.

In the first group, where color interlacing was present, when the fundus and optic discs were found normal in appearance or even when the disc showed the pallor of atrophy, it is strikingly notable that, although the patient entered the hospital under suspicion of having intracranial pressure due to a new-growth, it was frequently demonstrated later that such was not the case. The final diagnosis in these

cases may have been paraclonus multiplex, idiopathic epilepsy, osteoma of skull, labyrinthine vertigo, hyperthyroidism, arteriosclerosis, migraine, or pseudo brain-tumor. And still more strikingly negative were the cases found to have hypophyseal struma. The optic discs in these patients not only showed no evidence of intracranial pressure, but rather the pallor of optic atrophy or physiologic blocking of the optic nerve, frequently without the formation of an internal hydrocephalus which could dilate the sheath of Schwalbe. One might feel then that the explanation of color interlacing was not obvious on anatomic grounds, and turn to the observers. Here may be found a considerable variation. Some observers find color interlacing frequently and others do not. This is well shown in No. 28132, a case of hypophyseal struma on which four observers took fields before operation, with proportionate variation in result, and also the different observations of the same examiner, "A," are noted to vary on the same case.

In contrast to the first group, the second group shows many observations of no color interlacing, although the optic discs showed evidence of cerebral pressure, often not measurably elevated, but the presence of cerebral tumor or pressure was later confirmed in many of the group. Here also color interlacing may be found by one observer, and not by another observer under the same conditions. After operation color interlacing is rarely found, no matter what the condition of the optic disc may be. Before operation in several cases it was not found in the first field, but in the second field usually by a different observer.

In the small third group color interlacing was found always before operation, and the optic discs were edematous, but not measurably elevated. As a rule, only one field was taken before operation. In two cases, the third and the last, color interlacing persisted after the decompression. In three other cases it was not found after operation.

In the fourth group are found a large number of cases with measurable choked discs, but no color interlacing before operation, with the single exception of the first case, where the second field showed color interlacing. In this same case, and in two others in the group, color interlacing was noted after operation and measurable elevation was still present, but for the most part the field is recorded free from color interlacing after the decompression, no matter what grade of choked disc persists.

In the fifth group, with measurable choked discs, color interlacing was found sometimes in one eye only before operation. After operation, with measurable choked disc still present, nine cases are recorded with color interlacing present in one or both eyes, and nine cases with color interlacing absent in both eyes under the same conditions. Four other cases are variable in this respect, having fields which both do and do not show color interlacing. Two of these instances occurred with the same observer and two with different observers.

Considering the entire number of fields, presented in a manner as unbiased as possible, with no particular consideration of the fields beyond the indication as to the presence or absence of color interlacing, it would appear that, although there is some evidence that the presence of color interlacing bears a relation to intracranial pressure, there is even stronger evidence that personal equation on the part of the observer, as well as that of the patient, may go far in accounting for the results. But further insight into the question may be obtained by studying the character of the fields and color interlacing as found in the records. The papers dealing with the subject of color interlacing present cases and fields showing the best and most characteristic examples bearing on the question, so that a study of these typical cases will satisfactorily cover the ground.

DISCUSSION OF PREVIOUS PAPERS.

The proposition of color interlacing as an index of intracranial pressure rests largely, if not wholly, on the evidence submitted in two papers. The first of these* shows some of the very early fields taken in Professor Cushing's clinic at the Johns Hopkins Hospital. There is no mention of perimetric technic beyond the indication of the character of colors used. In over half of the fields the visual acuity is not given, and in others where it was given, or where it has since been looked up, it was so poor, sometimes less than 8/200, that poor fixation alone could account for the interlacing found in markedly contracted color-fields. In one of these fields without a visual acuity (Fig. 2, Surg. No. 15893) the form field is shown running for some distance inside both color-fields to the same size of disc. It might be thought that it was not meant for the form field, but rather for the field for white. However, it is expressly stated in the legend and in the text that it is the form field, and that it runs inside the color-fields. This would seem to me to be by definition an error, since where color can be seen, motion of the same size of object must also be detected. Certainly I have never seen anything at all similar to this condition which is given not as an isolated instance, but as a group characteristic. It must be noted, however, that this was one of the very earliest fields, and some confusion may have existed.

But still more difficult for us to understand in the light of our own experience is the peculiar behavior of the field for red in a large percentage of the fields showing color interlacing before but not after operation. This peculiarity occurs in two out of four such cases in the first paper, and all except possibly one of seven such cases in the second paper.† At

* Bordley and Cushing: Archives of Ophthalmology, 1909, xxxviii, No. 5.

† Cushing and Hener: Jour. Amer. Med. Assoc., 1911, lvii, 200.

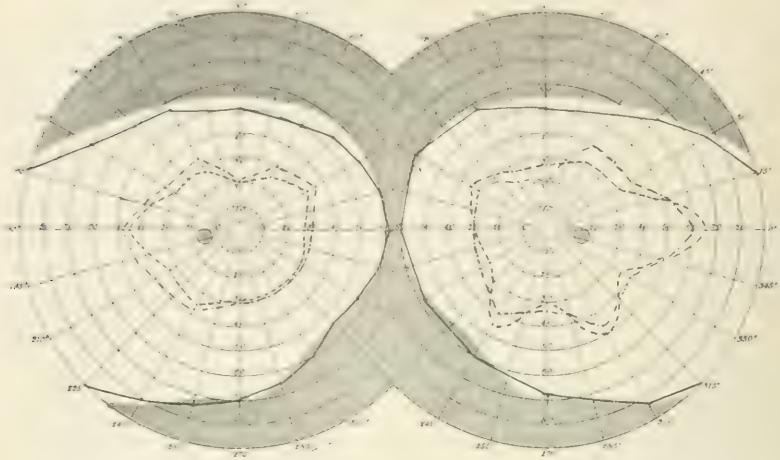


Fig. 7 A.—(Case IV) Chart before operation, showing color interlacing in the right field. Blue—.—.; Red----

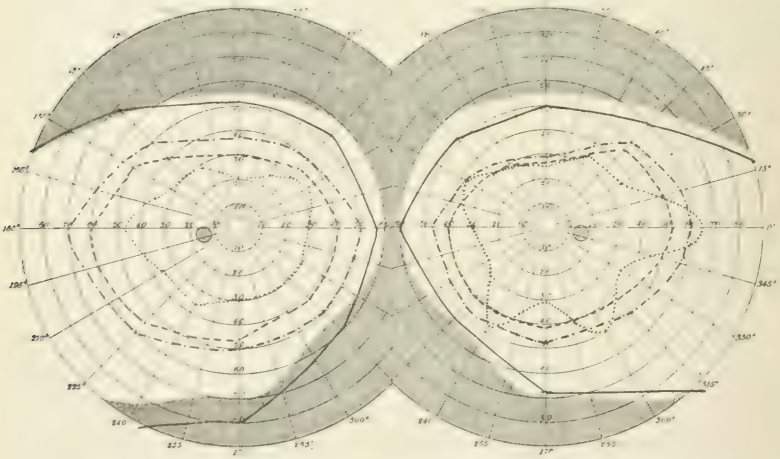


Fig. 7 B.—(Case IV) Chart after operation, with absence of color interlacing. The dotted line represents the red field before operation (Fig. 7A), superimposed to illustrate the shrinkage after operation of the red field at the points of interlacing.

the points of interlacing, before operation only, the reading for red may even be recorded not only wider than the normal average for 5 mm. discs, but, what is more notable, considerably wider at these points than is found after decompression, when all other measurements except those for red in the interlaced regions have shown great improvement. In other words, the field for red has contracted after decompression, particularly at the points of interlacing readings, while practically all the other readings in the field, both for color and form, have expanded. This phenomenon is well shown in Fig. 7 (A and B), which is a reproduction of the fields for Case IV in the second paper. In the left eye no color interlacing was found before operation, and marked improvement is noted in all the readings after operation, while in the right eye, where color interlacing was found, the readings for red contracted after operation at the points where interlacing took place, although all the other readings for color showed considerable increase. This is shown more clearly in any of the cases by transposing the red field before operation on to the red field after operation for comparison, as has been done in Fig. 7 (B). It will then be noted that, while the blue field almost always improves after operation, the red field may contract at points of interlacing, or, in less marked cases than the one shown, may remain stationary while the rest of the field improves. The amount of shrinkage of the red field at these points may be marked. Thus, in the right eye of Case IX, Figs. 15 and 16, it is almost 20 degrees.

Color interlacing is not accounted for in the original papers so much by this phenomenon as by the occurrence of peripheral scotoma for blue. Indeed, the behavior of the red field is not discussed at all, but if the peripheral scotomatous damage does occur for blue, it does not seem obvious to me why the vision for red should be improved in those same damaged regions to so marked a degree. In my own experience I must admit that I have not seen such conditions

in brain cases, but rather have the peripheral damage to the color-fields before decompression and the recovery afterward been seen to proceed much more uniformly. If anything, the color-fields have suffered more in the inverse order of their size, that is, I have more commonly seen peripheral blue vision preserved to the last and green vision suffer first, with later failure for red. Likewise, the last to go has commonly been the first to return after operation. Exceptions to this order have usually been in cases of centrally situated scotomata, either of syphilitic or other toxic etiology, or in cases not of cerebral tumor origin. Altogether we have felt that the explanation of color interlacing as it has been found in the past is not so much to be found in the occurrence of peripheral scotomata for a particular color, as in the psychology of field taking, and particularly in the peculiar record of the red field at the points of so-called interlacing.

FINDINGS IN THE PETER BENT BRIGHAM CLINIC.

Regarding cases having choked disc or suspected intracranial pressure, examined in Professor Cushing's clinic at the Peter Bent Brigham Hospital, there is not a great deal to say concerning color interlacing beyond the fact that it has not been found except once by an intern taking his first field on a patient who had no pressure manifestations. The fields for red and blue have been seen very close together, even in contact, and bizarre readings have been obtained on very sick patients or patients suffering intense headache, but these conditions always realigned themselves when the patient was carefully examined in an interval when free, or almost free, from pain and in a comfortable position. Apparently, color interlacing will be more or less present when these and other factors are not controlled. But even in these cases, if one is willing to force the patient, even in the presence of pain, to make a number of observations as accurately as can be done

under the circumstances, the average will show an absence of color interlacing.

It is interesting to note that in the few patients here who have had hysteric manifestations color interlacing has not been found. However, anything may apparently happen in hysteria, and it is quite possible that such a patient might have a liking or predilection for red or hate to recognize blue to such an extent that color interlacing or inversion would be obtained not so much from a functional as a mental derangement.

CONCLUSIONS.

1. For the detection of early pressure effects on the optic tract examination of the fields for the smallest visual angles gives the most reliable results.

2. Color interlacing is not a reliable test for cerebral pressure.

3. Most of the color interlacing found in the past in brain cases may be accounted for by a variety of variables, largely psychologic and physical.

4. Perimetry on semi-reclining patients by artificial illumination imitating daylight has been found practical.

I wish to extend my most grateful thanks to Professor Harvey Cushing for his kind generosity in allowing me to present this particular phase of his cases.

I

OPTIC DISCS NORMAL OR WITH ATROPHIC PALLOR, BUT OFTEN SHOWING COLOR INTERLACING (42 CASES).

SURG. NO.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINER
24923	Before	Paraclonus multiplex	O.U. No C.D.	O.U. C.I.	1 f	H
26270	Before	Epilepsy	O.D. No C.D.	O.D. C.I.		
			O. S. No C.D.	O. S. No C.I.	1 f	H
23887	Before	Epilepsy	O.U. No C.D.	O.U. C.I.	2 f	F
	After		O.U. No C.D.	O.U. No C.I.	1 f	F
20071	Before	Epilepsy	O.U. No C.D.	O.U. C.I.	1 f	D
24408	Before	Epilepsy	O.U. No C.D.	O.D. C.I.		
				O. S. contact	1 f	F
	After		O.U. No C.D.	O.D. No C.I.		
				O. S. C.I.	2 f	F
26747	Before	Osteoma skull	O.U. No C.D.	O.U. C.I.	1 f	H
28134	Before	Epilepsy	O.U. No C.D.	O.U. C.I.	1 f	I
28219	Before	Hypophyseal struma	O.U. No C.D. optic atrophy	O.D. No C.I. O. S. C.I.	1 f	F
22665	Before	Hypophyseal struma	O.U. No C.D. optic atrophy	O.D. C.I. O. S. nil	1 f	H
27580	Before	Labyrinthine vertigo	O.U. No C.D.	O.U. C.I.	1 f	J
23407	Before	Enceph. malacia	O.U. No C.D.	O.U. C.I.	1 f	F
26933	Before	Epilepsy	O.U. No C.D.	O.U. C.I.	1 f	H
25806	Before	Hypophyseal struma	O.U. No C.D. optic atrophy	O.D. nil O. S. C.I.	1 f	H
	After		O.U. No C.D.	O.D. nil O. S. No C.I.	1 f	H
28132	Before	Hypophyseal struma	O.U. No C.D.	O.U. C.I.	1 f	A
	Before		O.U. No C.D.	O.U. No C.I.	3 f	E
	Before		O.U. No C.D.	O.U. C.I.	1 f	I
	Before		O.U. No C.D.	O.U. No C.I.	1 f	G
	Before		O.U. No C.D.	O.D. C.I. O. S. No C.I.	1 f	A
24104	After	Osteoma skull	O.U. No C.D.	O.U. C.I.	1 f	F
27104	Before	Dyspituitarism	O.U. No C.D.	O.U. C.I.	2 f	H
Readmission			O.U. No C.D.	O.U. No C.I.	2 f	I
27414	Before	Epilepsy	O.U. No C.D.	O.D. No C.I. O. S. C.I.	1 f	J
27397	Before	Epilepsy	O.U. No C.D.	O.D. No C.I. O. S. C.I.	1 f	H
	After		O.U. No C.D.	O.U. No C.I.	1 f	H
28708	Before	Hyperthyroidism	O.U. No C.D.	O.D. No C.I. O. S. C.I.	1 f	G
	Before	Hyperthyroidism	O.U. No C.D.	O.U. No C.I.	2 f	G
25694	Before	Hypopituitarism	O.U. No C.D.	O.U. C.I.	1 f	H
	Before		O.U. No C.D.	O.U. No C.I.	1 f	H
27238	Before	Hypopituitarism	O.U. No C.D.	O.U. C.I.	1 f	I
Readmission			O.U. No C.D.	O.U. No C.I.	1 f	E
			O.U. No C.D.	O.U. No C.I.	1 f	G
24165	Before	Cereb. art. sclerosis	O.U. No C.D.	O.U. C.I.	1 f	F
24978	Before	Migraine	O.U. No C.D.	O.U. C.I.	1 f	H
26921	Before	Migraine	O.U. No C.D.	O.U. C.I.	1 f	H
23765	Before	Brain tumor (?)	O.U. No C.D.	O.U. C.I.	1 f	D
28491	Before	Epilepsy	O.U. No C.D.	O.D. C.I. O. S. No C.I.	1 f	J
27798	Before	Hypophyseal tumor	O.U. No C.D. optic atrophy	O.U. C.I.	1 f	J
	After		O.U. No C.D. optic atrophy	O.U. No C.I.	1 f	J

OPTIC DISCS NORMAL OR WITH ATROPHIC PALLOR, BUT OFTEN SHOWING COLOR INTERLACING (42 CASES).—(*Continued.*)

SURG. No.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINER
26950	Before	Brain tumor (?)	O.U. No C.D.	O.U. C.I.	1 f	H
	After		O.U. No C.D.	O.U. No C.I.	1 f	H
26250	Before	Hypophyseal tumor	O.U. No C.D. atrophic	O.D. No C.I. O. S. C.I.	1 f	N
	Before		O.U. No C.D. atrophic	O.U. No C.I.	1 f	N
	Before and after		O.U. No C.D. atrophic	O.U. No C.I.	1 f	H
23150	Before	Syphiloma	O.U. No C.D.	O.U. C.I.	1 f	F
	After		O.U. No C.D.	O.U. No C.I.	1 f	F
25736	Before	Chronic arachnoiditis	O.U. No C.D.	O.U. C.I.	1 f	H
	After		O.U. same	O.U. No C.I.	1 f	H
25943	Before	Headache (B.T.?)	O.U. No C.D.	O.U. C.I.	1 f	F
	Before		O.U. No C.D.	O.U. No C.I.	1 f	F
28498	Before	Brain tumor (?)	O.U. No C.D. prim. op. atrophy	O.D. Color inversion O. S. No C.I.	1 f	E
25396	Before	Brain tumor	O.U. No C.D.	O.U. C.I.	1 f	H
	Before		O.U. No C.D.	O.U. C.I.	1 f	F
	After		O.U. No C.D.	O.U. No C.I.	2 f	H
26204	Before	Brain tumor (?)	O.U. No C.D.	O.D. No C.I. O. S. C.I.	1 f	H
	After		O.U. No C.D.	O.U. C.I.	2 f	H
20214	Before	Brain tumor	O.U. No C.D.	O.U. C.I.	1 f	D
26622	Before	Brain tumor (?)	O.U. No C.D.	O.U. C.I.	1 f	J
	After		O.U. No C.D.	O.D. C.I. O. S. No C.I.	1 f	H
26387	Before	Brain tumor (?)	O.U. No C.D.	O.U. C.I.	1 f	H
28485	Before	Hypophyseal tumor	O.U. No C.D. optic atrophy	O.U. C.I.	1 f	H
	Before		O.U. No C.D. optic atrophy	O.U. No C.I.	1 f	I
19990	Before	Epilepsy	O.U. No C.D. optic atrophy	O.D. Red vision nil O. S. C.I.	1 f	D
	Before		O.U. No C.D. optic atrophy	O.U. Red vision nil, blue preserved.	1 f	D
27955	Before	Hypophyseal struma	No C.D. atrophic	O.U. No C.I.	2 f	G & F
28266 (Readmission)	After	Hypophyseal struma	No C.D. atrophic	O.U. C.I.	1 f	I
29811 (Readmission)			No C.D. atrophic	O.U. No C.D.	1 f	E
25389	Before	Cereb. art. sclerosis	No C.D.	O.U. C.I.	1 f	H
	After		No C.D.	O.D. C.I. O. S. No C.I.	1 f	H

II

OPTIC DISCS EDEMATOUS BUT NOT MEASURABLY ELEVATED, BUT OFTEN SHOWING NO COLOR INTERLACING (16 CASES).

SURG. No.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINER
29601	Before and after	Temp. lobe tumor	O.U. discs edematous	O.U. No C.I.	3 f	E
25463	Before	Brain tumor	O.U. C.D. low grade	O.U. No C.I.	2 f	H
29181	Before	Epilepsy	O.U. edematous	O.U. No C.I.	1 f	H
	Before		O.U. edematous	O.U. C.I.	1 f	A
	Before		O.U. edematous	O.U. No C.I.	1 f	E
25645	Before	Brain tumor	O.U. edematous	O.U. No C.I.	1 f	D
	Before	Brain tumor	O.U. edematous	O.U. C.I.	1 f	H
	After		O.U. edematous	O.U. No C.I.	1 f	H
23689	Before	Brain tumor (?)	O.U. edematous	O.U. No C.I.	1 f	H
27322	Before	Brain tumor	O.U. edematous	O.U. No C.I.	1 f	I
26369 (Readmission)		Brain tumor	O.U. 2 D	O.U. C.I.	1 f	H
	After		O.U. No C.D.	O.U. No C.I.	1 f	H
25600	Before	Brain tumor	O.U. edematous secondary changes	O.U. No C.I.	1 f	H
26076	Before	Osteoma	O.U. C.D. low grade	O.U. No C.I.	1 f	F
21674	Before	Brain tumor	O.U. edematous	O.U. No C.I.	1 f	E
26068	Before	Hypophyseal tumor	O.U. C.D. low grade	O.U. No C.I.	1 f	H
25053	Before	Art. sclerosis	O.U. edematous	O.U. No C.I.	1 f	H
26454	Before	Brain tumor	O.U. C.D. not measurable	O.U. No C.I.	1 f	C
	Before		O.U. C.D. not measurable	O.U. C.I.	1 f	H
	Before		O.U. C.D. not measurable	O.U. contact	1 f	H
	After		O.U. C.D. not measurable	O.U. No C.I.	1 f	H
	After		O.U. C.D. 1 D	O.U. No C.I.	1 f	I
	After		O.U. disc elevated 1 D	O.U. No C.I.	1 f	I
26980	Before	Brain tumor, epilepsy	O.U. C.D. edematous, not measurable	O.D. No C.I. O. S. C.I.	1 f	H
	Before	Brain tumor, epilepsy	O.U. C.D. edematous, not measurable	O.U. C.I.	1 f	H
24942	Before	Brain tumor	O.U. edematous low grade C.D.	O.U. No C.I.	1 f	H
25435 (Readmission)		Int. hydrocephalus	O.U. edematous low grade C.D.	O.U. slight C.I.	1 f	H
	After	Int. hydrocephalus	O.U. edematous	O.U. No C.I.	1 f	H
24882	Before	Cerebral edema, pseudo tumor, hysteria	O.U. edematous veins engorged	O.U. No C.I.	1 f	H
29787	Before	Brain tumor	O.U. very edematous, veins engorged and tortuous	O.U. No C.I.	4 f	E

III

OPTIC DISC NOT MEASURABLY ELEVATED, BUT COLOR INTERLACING PRESENT (7 CASES).

SURG. No.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINER
24033	Before	Brain tumor	Receded (?) C.D.	O.U. C.I.	1 f	F
23604	Before	Brain tumor (?)	O.U. edematous	O.U. C.I.	1 f	F
	After	Uncinate fits	O.U. same	O.U. No C.I.	1 f	F
19853	Before	Brain tumor (?)	O.U. C.D. not measurable	O.U. C.I.	1 f	D
	After		No C.D. O. U.	O.U. C.I.	1 f	D
19636 (Readmission)			O.U. C.D. not measurable	O.U. C.I.	1 f	D
28674	Before	Cerebellar tumor	O.U. No C.D. hyperemic	O.U. C.I.	1 f	G
	After		O.U. No C.D.	O.U. No C.I.	1 f	G
23482	After	Brain tumor	O.U. sl. edema	O.U. C.I.	1 f	D
	After		O.U. disc color	O.U. No C.I.	1 f	A
23779	Before	Acromegaly	O.U. No C.D. sl. edema	O.D. contact O. S. C.I.	1 f	F
26847	Before	Brain tumor	O.U. No C.D. O. S. edematous	O.U. C.I.	2 f	H
	After		O.U. elevated 2 D	O. S. C.I. O.D. No C.I.	1 f	H

IV

MEASURABLE CHOKED DISC WITHOUT COLOR INTERLACING (41 CASES).

SURG. No.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINER
24852	Before	Brain tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	K
	Before		O.U. C.D. 2 D	O.U. C.I.	1 f	H
	After		O.U. C.D. 1½ D	O.U. C.I.	1 f	H
27675	Before	Hypophyseal tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	J
	After		O.U. C.D. 1 D	O.U. No C.I.	2 f	J
23725	Before	Hypophyseal tumor	O.U. C.D. 1 D	O.U. No C.I.	1 f	F
24921	Before	Brain tumor	O.U. C.D. 3 D	O.U. No C.I.	1 f	F
27679	Before	Cerebellar tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	I
29771	Before	Brain tumor	O.U. C.D. 4 D	O.U. No C.I.	2 f	E
	After		O.U. C.D. 2 D	O.U. No C.I.	2 f	E
25117	Before	Brain tumor	O.U. C.D. 2½ D	O.U. No C.I.	2 f	H
27733 (Readmission)	After		O.D. C.D. 1 D O. S. C.D. 1½ D	O.U. No C.I.	2 f	J
28125	Before	Cerebellar tumor	O.U. C.D. 4 D	O.U. contact	1 f	I
29920 (Readmission for relief of pressure)	After		O.U. C.D. 2 D	O.U. No C.I.	2 f	E
26651	Before	Brain tumor	O.D. C.D. 2 D O. S. C.D. 1 D	O.U. No C.I.	1 f	H
25832	After	Brain tumor	O.U. C.D. 1 D	O.U. No C.I.	1 f	F

MEASURABLE CHOKED DISC WITHOUT COLOR INTERLACING (41 CASES).—*(Continued.)*

SURG. NO.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINED
22691	After	Brain tumor	O.U. C.D. 1 D	O.U. No C.I.	1 f	D
	After		O.U. C.D. 1 D	O.U. C.I.	1 f	D
27788	Before and after	Cerebellar tumor	O.U. C.D. 2½ D	O.U. No C.I.	2 f	I
28986	After	Hypophyseal tumor	O.U. C.D. 2 D receding	O.U. No C.I.	2 f	G
	After		O.U. C.D. 1 D	O.U. No C.I.	7 f	E
29395	Before	Hypophyseal tumor	O.U. C.D. 1 D	O.U. No C.I.	1 f	C
	After		O.U. C.D. 1 D	O.U. No C.I.	2 f	E
26194	Before	Brain tumor	O.U. C.D. 3 D	O.U. No C.I.	2 f	H
29935	Before	Brain tumor	O.D. C.D. 3 D O.S. C.D. 2 D	O.U. No C.I.	1 f	E
	After		O.U. C.D. 1 D	O.U. No C.I.	1 f	E
29289	Before	Hypophyseal tumor	O.U. C.D. 2½ D	O.U. No C.I.	1 f	E
27885	Before	Brain tumor	O.U. C.D. 3 D	O.U. No C.I.	1 f	J
29921	Before	Brain tumor	O.U. C.D. 3½ D	O.U. No C.I.	2 f	E
27300	Before	Brain tumor	O.U. C.D. 1 D	O.U. No C.I.	2 f	H
19941	Before	Brain tumor	O.U. C.D. 5 D	O.U. No C.I.	1 f	D
	After		O.U. C.D. 1½ D	O.U. C.I.	1 f	D
28096	Before	Osteoma skull	O.D. C.D. 4 D O. S. C.D. 7 D	O.U. No C.I.	1 f	I
	After		O.U. C.D. 2 D	O.U. No C.I.	3 f	I
29903	Before	Cerebellar tumor	O.U. C.D. 6 D	O.U. No C.I.	2 f	E
27686	Before	Brain tumor	O.U. C.D. 3½ D	O.U. No C.I.	1 f	I
29996	Before	Brain tumor	O.U. C.D. 3 D	O.U. No C.I.	1 f	E
	After		O.U. C.D. 2 D	O.U. No C.I.	1 f	C
	After		O.U. C.D. 2 D	O.U. No C.I.	1 f	E
28861	Before	Cerebellar tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	E
30210	Before	Brain tumor	O.U. C.D. 3 D	O.U. No C.I.	2 f	C
	After		O.U. C.D. 2 D	O.U. No C.I.	3 f	E
28084	Before	Brain tumor	O.U. C.D. 4 D	O.U. No C.I.	1 f	J
29248	Before	Brain tumor	O.U. C.D. 5 D	O.U. No C.I.	2 f	E
28332	Before	Cerebellar tumor	O.U. C.D. 3½ D	O.U. No C.I.	3 f	I
28595	Before	Cereb. syphilis	O.D. No C.D. O. S. C.D. 1½ D	O.U. No C.I.	1 f	E
	Before		O. S. C.D. 1½ D	O.U. No C.I.	2 f	G
	Before		O. S. C.D. 1½ D	O.U. No C.I.	1 f	B
28828	Before	Specific neuro-retinitis	O.U. C.D. 3 D	O.U. No C.I.	1 f	G
29162	Before	Brain tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	E
28981	Before	Brain tumor	O.U. C.D. 3 D	O.U. No C.I.	1 f	L
	Before		O.U. C.D. 3 D	O.D. No C.I. O.S. C.I.	1 f	X
29813	Before	Brain tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	E
29128	Before	Brain tumor	O.U. C.D. 7 D	O.U. No C.I.	1 f	E
28681	Before	Brain tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	G
29335	Before	Cerebellar tumor	O.U. C.D. 2½ D	O.U. No C.I.	1 f	E
27554	After	Brain tumor	O.U. C.D. 1 D	O.U. No C.I.	1 f	I
	After		O.U. C.D. 1 D	O.U. No C.I.	1 f	I
25224	Before	Brain tumor	O.U. C.D. 4 D	O.U. No C.I.	1 f	H

V

MEASURABLE CHOKED DISC WITH COLOR INTERLACING IN ONE OR BOTH EYES (37 CASES).

SURG. No.	OPERATION	DIAGNOSIS	FUNDI	FIELD	NUMBER	EXAMINER
27345	Before	Brain tumor	O.U. C.D. 3 D	O.U. C.I.	1 f	J
27513	Before	Brain tumor	O.U. C.D. 5½ D	O.U. C.I.	1 f	I
27186	Before	Brain tumor	O.U. C.D. 3½ D	O.U. C.I.	1 f	H
27619	Before	Brain tumor	O.U. C.D. 3½ D	O.U. C.I.	1 f	Z
	After	Brain tumor	O.U. C.D. 2 D	O.U. No C.I.	1 f	J
27700	Before	Cerebellopontine tumor	O.U. C.D. 4 D	O.U. C.I.	1 f	I
28263	Before	Brain tumor	O.U. C.D. 2 D	O.U. C.I.	1 f	I
27149	Before	Brain tumor	O.U. C.D. 6 D	O.U. C.I.	1 f	H
21654	Before	Brain tumor	O.D. C.D. 6 D	O.U. C.I.	1 f	D
			O.S. C.D. 3 D			
22488	Before	Brain tumor	O.U. C.D. 3 D	O.U. C.I.	1 f	D
22551	Before	Brain tumor	O.U. C.D. 6 D	O.U. C.I.	1 f	D
23479	Before	Brain tumor	O.D. C.D. 2 D	O.U. C.I.	1 f	F
			O.S. No C.D.			
24137	Before	Brain tumor	O.U. C.D. 3 D	O.U. C.I.	1 f	H
	After	Brain tumor	O.U. C.D. 2 D	O.U. C.I.	1 f	H
24307	Before	Brain tumor	O.U. C.D. 6 D	O.U. C.I.	1 f	F
24803	After	Brain abscess	O.U. C.D. 3 D	O.U. C.I.	2 f	H
27075	Before	Brain tumor	O.U. C.D. 7 D	O.U. C.I.	2 f	H
27131	Before	Brain tumor	O.U. C.D. 4½ D	O.U. C.I.	2 f	I
	After		O.U. C.D. 3 D	O.U. No C.I.	1 f	I
27619	Before	Hypophyseal tumor	O.U. C.D. 3 D	O.U. No C.I.	1 f	H
	Before		O.U. papillitis	O.U. C.I.	1 f	Y
	After		O.U. papillitis	O.U. C.I.	1 f	J
26390	Before	Brain tumor	O.U. C.D. 4 D	O.U. C.I.	1 f	H
	After	Brain tumor	O.U. C.D. 1 D	O.U. No C.I.	1 f	H
23857	Before	Brain tumor	O.D. C.D. 1 D	O.D. C.I.		
			O.S. C.D. 1½ D	O.S. No C.I.	2 f	F
	After		O.U. No C.D.	O.U. contact	1 f	F
26252	Before	Brain tumor	O.U. C.D. 4 D	O.U. C.I.	1 f	H
	After		O.U. C.D. 3 D	O.U. No C.I.	1 f	H
24778	Before	Brain tumor	O.U. C.D. 7 D	O.U. C.I.	2 f	H
	After		O.U. C.D. 5 D	O.D. C.I.		
				O.S. No C.D.	1 f	H
24398	Before	Brain tumor	O.U. C.D. 5 D	O.D. No C.I.		
				O.S. C.I.	1 f	J
23559	Before	Angioma meninges	O.U. C.D. 1 D	O.U. C.I.	1 f	F
	After		O.U. C.D. ½ D	O.U. No C.I.	1 f	F
23551	Before	Brain tumor	O.D. C.D. 3 D	O.D. No C.I.		
			O.S. C.D. 4 D	O.S. C.I.	1 f	F
	Before		O.U. C.D. 3 D	O.D. C.I.		
			O.S. C.D. 4 D	O.S. No C.I.	1 f	D
	After		O.U. C.D. 2 D	O.U. No C.I.	2 f	F
	After		O.U. C.D. 2 D	O.U. No C.I.	2 f	G
	After		O.U. C.D. 1 D	O.U. No C.I.	1 f	E
27399	After	Brain tumor	O.U. C.D. 2½ D	O.U. C.I.	1 f	I
27884	Before	Cerebellar tumor	O.D. C.D. 4 D	O.D. C.I.	1 f	I
	After		O.S. C.D. 5 D	O.S. no colors		
			O.D. C.D. 2 D	O.U. No C.I.	1 f	I
			O.S. C.D. 3 D			
25790	Before	Brain tumor	O.U. C.D. 5 D	O.U. C.I.	1 f	H
	After		O.U. C.D. 2 D	O.U. No C.I.	2 f	H
24920	Before	Brain tumor	O.D. C.D. 8 D	O.D. C.I.		
			O.S. C.D. 6 D	O.S. No C.I.	1 f	H
25339	Before	Brain tumor	O.U. C.D. 2 D	O.U. C.I.	1 f	H
	After		O.U. C.D. 1 D	O.U. No C.I.	2 f	H
24208	Before	Brain tumor	O.U. C.D. 2 D	O.D. No C.I.		
				O.D. C.I.	1 f	D
	After		O.U. C.D. 1½ D	O.D. C.I.		
				O.S. contact	1 f	F

MEASURABLE CHOKED DISC WITH COLOR INTERLACING IN ONE OR BOTH EYES.—(*Continued.*)

SURG. NO.	OPERATION	DIAGNOSIS	FUNDI	FIELDS	NUMBER	EXAMINER
22602	Before	Brain tumor	O.U. C.D. 4 D	O.U. C.I.	1 f	D
	After		O.U. C.D. 3 D	O.U. No C.I.	1 f	D
	After		O.U. C.D. 2 D	O.D. C.I.		
				O.S. No C.I.	1 f	D
26644	Before	Brain tumor	O.D. C.D. 1½ D	O.D. C.I.		
			O.S. C.D. 2 D	O.S. No C.I.	1 f	H
25292	Before	Brain tumor	O.U. C.D. 3 D	O.U. C.I.	1 f	H
	After		O.U. C.D. 2 D	O.U. No C.I.	2 f	H
	After		O.U. C.D. 2 D	O.D. No C.I.		
				O.S. C.I.	1 f	H
22671	Before	Brain tumor	O.U. C.D. 3 D	O.U. C.I.	1 f	D
	After		O.U. C.D. 2½ D	O.U. No C.I.	1 f	D
23385 (Readmission)	After		O.U. No C.D.	O.U. C.I.	1 f	F
	After		O.U. No C.D.	O.U. No C.I.	1 f	H
28535 (Readmission)	After		O.U. No C.D.	O.U. No C.I.	2 f	H
	After		O.U. C.D. 2 D	O.U. No C.I.	1 f	E
24119	Before	Brain tumor	O.U. C.D. 5 D	O.U. C.I.	1 f	F
	After		O.D. C.D. 3 D	O.D. No C.I.		
			O.S. C.D. 2 D	O.S. C.I.	2 f	F
28519	Before	Brain tumor	O.U. C.D. 3 D	O.D. No C.I.		
				O.S. C.I.	1 f	J
	After		O.U. C.D. 1 D	O.U. No C.I.	1 f	C
27583	Before	Brain tumor	O.U. C.D. 2½ D	O.U. C.I.	1 f	I
	Before		O.U. C.D. 3 D	O.D. C.I.		
				O.S. No C.I.	1 f	I
	After		O.U. C.D. 1 D	O.U. No C.I.	1 f	I

A DISCUSSION OF SOME NEWER PRINCIPLES IN DEALING WITH UNCOMPLICATED CATARACT.*

DERRICK T. VAIL, M.D.,
Cincinnati, Ohio.

In entering a plea for the consideration of so-called "newer principles" in the cataract problem, it seems necessary to state at the onset that this term is used in a comparative sense.

It is not claimed that cataract surgeons have agreed on a code of unalterable principles, which are accepted *ex cathedra* by the rank and file of operators. On the contrary, there is

* Candidate's thesis for membership accepted by the Committee on Theses.

a bewildering array of postulates propounded in such a bewildering mass of literature on the subject of cataract surgery that it may be truly said that there are no "established principles" in the sense of dogmas or creeds.

Generally speaking, however, there are certain guiding principles taught in most of the college class-rooms of ophthalmic students and observed in actual practice among the majority of the leading ophthalmologists of the world. These we shall call the "established principles," and they furnish the impulse of our argument in favor of other so-called "newer principles"; newer, in the sense that they are founded on more recent advances and are not commonly known or practised.

In bringing to your attention some of these newer principles the writer does not present any original methods, but simply collects those which are scattered in many standard text-books and classic monographs on the cataract subject by authors of renown. They are here collected and presented for the sake of study, comparison, and discussion.

The subject naturally divides itself into two headings, viz.: (*a*) *Major principles*, or those dealing with the economic problem of vision; (*b*) *minor principles*, or those dealing with methods.

Of the major principles, we shall confine ourselves to the discussion of but two, viz.:

I. That preliminary iridectomy is a beneficial measure in certain forms of slowly forming or stationary cataract, and

II. That the patient must wait until his cataract is ripe, or nearly so, before the operation is advisable.

These two major principles are so linked together that it is difficult to discuss them separately, for the former is sustained or justified by the latter, and the most successful outcome of the latter is, in the minds of many, dependent on the former. Nevertheless, it seems best to discuss them, each on its own merits.

I. The Question of Preliminary Iridectomy.—It is the custom in the practice of many, if not the majority of, cataract operators to perform preliminary iridectomy in certain forms of immature cataract, particularly those in which the cataracts are slowly forming or stationary. The argument in brief is that, from the patient's standpoint, this surgical measure will increase his vision while waiting for cataract maturity and will permit him to go about with reasonable safety until the lens begins to become more opaque; and that, when the cataract does begin to increase, the period of incapacity is not prolonged over many months until it may be ripe enough to extract by the established method.

From the surgeon's standpoint the operation proposed is fraught with little danger, is short, painless, trains the patient not to fear the surgeon, and holds him until the time when his cataract is finally deemed ripe enough for extraction; moreover, that the steps of the final operation are thereby shortened, made easier, and more certainly successful. It is interpreted by the patient to be a better assurance that his sight will be restored ultimately.

The present writer followed this principle for many years, particularly in dealing with the more intelligent class of patients. He has abandoned it entirely during the past five years, has nothing to regret and no desire to return to it, for he is convinced that it is founded on surgical sophistry, or in some cases timidity, rather than on sound judgment and the patient's best welfare.

There are valid arguments against preliminary iridectomy in these cases, the strongest being that, in the light of modern knowledge, verified from many sources, cataract can be easily removed at any stage of ripening, and there is call for extraction the moment the question of preliminary iridectomy comes up. But leaving the question of proposed extraction out of the argument, there are good reasons to decry the preliminary iridectomy:

First: It is an operation where the eyeball is opened, and as such does contain an element of risk, however slight.

Second: The result is often disappointing to the patient, for whatever the improvement in gross or quantitative vision, there is little or no restoration of reading or qualitative vision.

Third: The preliminary iridectomy does not simplify the extraction operation in the hands of an expert; on the contrary, may seriously complicate it, as I have several times observed.

Fourth: One operation is better than two when the same result is obtainable.

Fifth: A preliminary iridectomy necessitates a scar in the limbus—a second scar in the same locality is objectionable.

As will be shown presently, preliminary iridectomy is never indicated in uncomplicated cataract, for when the vision is impaired to the degree that it would be justified according to the old teaching, the time has arrived for the actual extraction, regardless of the kind of cataract present.

Therefore I wish to maintain that the following principle is sound; viz.:

A preliminary iridectomy in immature uncomplicated cataract is unnecessary and needless, and is therefore never indicated.

II. As Regards the Old-established Principle that the Cataract must be Ripe, or Nearly So, Before Extraction.—It is the generally accepted dictum, enunciated by the majority of the world's highest authorities from the time of the illustrious founders of ophthalmic practice of over fifty years ago down to the present day, that the cataract must pass through the various stages of ripening, from the incipient to the ripe or nearly ripe stage, before the most favorable time for extraction has arrived. The period of waiting from the first symptoms until the cataract is deemed ideal for operation is rarely less than two years, and not infrequently five years and longer. Indeed, one of the most

common forms of cataract never gets ripe according to the accepted definition of "ripeness." During all that time the patient is required to go about half blind. It is rarely that he is contented to accept the opinion of the oculist of his choice on this matter of waiting, and so he seeks other counsel, only to find it is too true: he must wait.

The kindest, most conscientious, and most skilful surgeon, who follows and wishes to follow the advice of those who taught him his best lessons in the science and art of ophthalmic practice, needs must tell his patient "your cataract is not ripe enough; you must wait at least six months, perhaps two years," and this, too, while he is still in prime health and vigor and is splendid surgical material in that the most scrutinizing test fails to reveal anything else wrong with him, mentally or physically.

This principle is so firmly grounded in the minds of the vast majority of cataract surgeons that it amounts to a religion. The argument that the patients who come to them with immature cataracts seeking relief are entitled to have relief, and that it is their duty to furnish them that relief, does not seem to weigh against the terrors that arise in their minds when they think of operating on a man only half-blind with cataract. And so these unfortunate patients are given "placebo" drops, and put off from month to year, until what is gained in the imagination that the cataract is getting gradually ready for operation is lost in the actual fact that the prime vigor of bodily and mental health is deteriorating. Nothing so undermines the foundations of good health and reason as suspense and worry. The newer principle, which during the past five years I have followed in managing these cases of immature cataract, takes the above facts into consideration and for their full value, and the results achieved justify the faith that is in me. The statistical table at the end of this paper, showing the results achieved in 40 consecutive cases of uncomplicated immature cataract

operated upon by me during the past five years, furnishes the basis of that faith in the newer principle.

We are dealing with principles in this paper, and to those who for any reason stand out against the intracapsular method of extracting the immature cataract I wish to present evidence from some who have employed the capsulotomy operation in this stage of cataract.

Weeks, of New York, in a paper on "What Stage in the Development of Cataract, Particularly Senile, is the Most Suitable for its Removal by Extraction,"* states: "On the strength of these observations (experiments in the dead room on extraction of transparent lenses), in spite of the dictum 'hands off!' in reference to immature cataract, I began to remove the cataractous lens soon after it had reached a degree of opaqueness sufficient to interfere with useful vision, whatever the form of the cataract might be, in all cases in which such removal was particularly urgent, those being excepted in which the lenses were much swollen." He reports 25 successive cases of immature and zonular cataract on which he had operated, and he finishes his article with the following declaration: "When so done, the visual results are not much, if any, below those obtained after removal of mature cataract."

Fuchs† advises waiting until the cataract is ripe, but states: "The cataract may be extracted before it is fully ripe. Healing takes place with as good results as in ripe cataract.

Meller, speaking of capsule forceps,‡ states: "A large opening is made in the anterior capsule directly in the pupillary area. The result of this loss of capsule tissue is that *the capsule cannot produce optical disturbances later on. The remnants of cortex left behind are exposed to the action of the aqueous humor, with the result that, even in the operation for*

* "Cataract Extraction," Claiborne, New York, 1908, page 6.

† Text book, Am. edition, 1911, page 524.

‡ "Ophthalmic Surgery," English edition, 1908, p. 134.

unripe cataract or when a large amount of cataract-remnants are present, they usually undergo spontaneous absorption after the operation."

Roemer* states: "Vision is very often impaired by the nature and situation of the lenticular opacities, so that the patient is incapacitated for work before the cataract has become ripe, and we should not needlessly leave such an unfortunate person in the torture of uncertainty because of an antiquated clinical conception. In former years many patients passed away with their hope of restoration of sight to their eyes unfulfilled."

He states the newer principle in a nutshell when he adds: "*If the vision is impaired so that the patient is unable to work, and the prognosis is justified that vision can be adequately restored by extraction of the lens, the cataract is ready for operation.*"

His requisite for a good prognosis is merely that the retina is functionating, the eye is clean, and the patient in good health.

Smith, of India, who holds the world's record on numbers and, I believe, on results too, has no compromise to offer in dealing with this problem. With characteristic directness he states:† "*When the lens becomes so opaque that vision is no longer useful for ordinary practical purposes, it should be extracted in its capsule.*"

It is not the purpose of this paper to discuss the relative merits of the different operations for ridding the eye of immature cataract. There are four different methods of technic that I know of for the intracapsular extraction of cataract, and three at least for the extracapsular extraction. It may be that any of these seven methods, rightly carried out, will succeed in as large a proportion of cases as can be achieved by waiting until the cataract is ripe and then extracting after slitting the capsule with a cutting instrument.

* Text book of Ophthalmology, American translation, 1912, p. 226.

† Treatment of Cataract, 1910.

To those who hold to the old-established principle I will say, patients are finding out that relief from semi-blindness because of immature cataract can be had, and they are seeking that relief. I ask if you, in the prime of health, otherwise were to become incapacitated on account of immature cataract, would you be content to wait until you could barely count your fingers at one foot from your eyes before submitting to an operation?

The day has arrived when we must consider what has been done for others afflicted with nuclear or immature cataract, and it behooves us to adopt the newer principle and diligently seek the best and surest way of succeeding by that principle.

The argument above stated resolves itself into the following logic:

(*a*) A patient has immature cataract of one form or another. His vision is, we will say, 20/200; he can no longer read ordinary print. The fundus can still be seen and is normal. The eye is sound aside from the cataract. His general health is good.

(*b*) Immature cataract of any form can be successfully removed by several methods, and the results in large numbers compare favorably with the results of operation by the established method on mature cataracts.

(*c*) Therefore: Operate without waiting. It is humane, good surgery, and our bounden duty.

Of the *minor principles,* or those dealing with *methods,* we shall discuss briefly those affecting the following:

(*a*) Cleansing the eye for operation.
(*b*) Controlling the eyelids during the operation.
(*c*) Dealing with the capsule.
(*d*) Dealing with retained lens-substance.
(*e*) Time as a factor.
(*f*) Atropin.

It is taken for granted that most surgeons will maintain that the established technic used in dealing with each of the above list of methods is designed to carry out the principle that the best possible things are done to achieve the best results.

As my practice among the 40 cases of very immature cataract cited in the appendix of this paper has been different in principle from the established methods as concerns this list, and as I think the newer principles employed contributed largely to results achieved, I desire to state them for your consideration. I wish again to disclaim any originality in these matters, for each point was taken from the writing, teaching, and practice of others. I adopted them because they appealed to me as being sound and better than those ordinarily employed. In most of them I was drilled by my distinguished teacher, Colonel Smith.

(*a*) *As regards cleansing the eye for operation:* It is well known that anything which bruises the delicate epithelium of the palpebral or ocular conjunctiva just before operation is objectionable. The best protection against invading infections is a sound epithelial covering. It is therefore wrong to squeeze the lids to evacuate the Meibomian glands, for this cannot be done without insult to the delicate epithelium. Moreover, the thumb-nail or finger-pulp is not always above suspicion, and, furthermore, I am not convinced that the Meibomian secretion is infective; on the contrary, I am satisfied it is not so. After it has been extruded by the squeezing method, the newly secreted material is certainly no improvement on that which was expressed. Also it is not good practice, in my judgment, to use moist cotton sponges for swabbing the conjunctival sac prior to operation. The sponges may abrade the epithelium; at any rate they must be handled and squeezed out with the fingers, and there again the argument that the fingers are not above suspicion comes up.

It is also manifestly impossible to reach the culdesac with the stream of fluid from a douche-bottle, douche-bulb, or undine when the lids are everted and held apart in the usual way.

The folds of the fornices, especially of the upper, are still collapsed, and the opposing surfaces are in contact. The douche is therefore not effective in reaching their surfaces.

The newer principle I wish to mention takes cognizance of the above facts, and also of the fact that all that can be accomplished in the normal-appearing eye is to wash out the cells and mucus, which represents the normal shedding or excretion from the epithelium of the mucous membrane. Nature secretes the tears for that purpose in the functionating eye where the lids are constantly winking and assisting in carrying off this débris, but after operation the lids no longer wink, and this material already there had better be well washed out of the sac.

Hence the "newer principle"—no rubbing and no violence, but a full and effective douching with a large stream of warmed salt water, boric acid solution, or weak bichlorid solution, and this done while the eyelids are held away from the eyeballs on a proper speculum in such a manner that the folds of the fornices are no longer "folds" at all, but the entire sac is one smooth-walled pouch, inviting the swirl of fluid to wash away all detached cells, mucus, and germs.

The "Jullundur speculum" is the only one I know of that will hold the lids when they are lifted to the limit away from the eyeball. By making upward traction on the eyebrow at the same time that the lids are being hung well off the eyeball, the upper sac is thoroughly exposed for the douche stream, as can well be seen by peeping under.

Results: I have performed over 500 extractions since adopting the above technic, and have not had a single case of panophthalmitis.

Results by the established methods of cleansing the eye: According to Ring's compiled statistics of 1032 extractions

"by operators of recognized ability in America and Europe,"* panophthalmitis occurred in nearly 2 per cent. of the cases in combined extraction.

(*b*) *As to controlling the lids during the operation:* Much has been written on the use of specula, lid-hooks, blepharostats, retractors, etc. Each operator maintains that his method is the best and guards against accidents the most. A cataract surgeon has not graduated in the highest sense until he has devised a new speculum or lid retractor. I have recently published† an article illustrating mine. I shall not describe it here.

I saw Sir Anderson Critchett, in 1899, do a combined extraction on the left eye with his left hand, employing *only one instrument throughout*, viz., a Graefe knife. He dispensed entirely with the services of an assistant. I never saw a more beautiful result in the combined extraction. He held the lids apart with the fingers of his right hand. No speculum was used. Meller uses no speculum in cataract extraction.‡ He permits the assistant to simply hold the lids apart with his fingers.

Some operators use a speculum throughout, and there is no denying the fact that they rarely have trouble. Others will use nothing but the lid retractors, more or less modified after the old Desmarres pattern, and they too meet with good success. Still others use the speculum during the section and iridectomy, and remove it during extraction, holding the eyelids with the fingers, and others again will use the speculum until the stage of delivery and then employ lid-hooks or elevators.

So far as the "squeezers" are concerned, I believe one method is exactly as good or as bad as the others. None will prevent the lens shooting out when the section is completed if the patient squeezes.

* Claiborne, "Cataract Extraction," p. 41.

† Ophthalmic Record, May, 1915.

‡ Ophthalmic Surgery, 1911.

The purpose and plan, therefore, of the speculum, retractor, or what-not is to facilitate the operation and guard against vitreous prolapse during the actual delivery. Vitreous escape is an inherent risk in every cataract extraction.

The principle I wish to urge here is that the eyelids, during actual delivery of the lens, should be held entirely off the eyeball and hung like a tent in order to take all weight and spasm off the eyeball and thus prevent as much as possible vitreous escaping during pressure to deliver the lens. By this method the amount of vitreous that need be lost in any case is very small—certainly never as much as by the old methods.

(*c*) *As to dealing with the capsule:* Many have accepted it as a guiding principle that the lens should be delivered in its capsule, and so several methods of technic to accomplish the *radical extraction of cataract* have been devised. I firmly believe that the time will come when this will be the generally accepted method of dealing with uncomplicated cataract. But since it is the anterior capsule that causes so much mischief and disappointment when left within the eye, it is certainly necessary, in doing the capsulotomy operation, to remove all or as much of it as possible by means of good capsule forceps.

The cystitome should be relegated to the museum of antiquated surgical instruments.

(*d*) *As to dealing with retained lens substance:* If the cataract is extracted in its capsule, the question of "retained lens substance" is answered at once. But in case the anterior capsule is removed in whole or in part, the question at once comes up, what is the best method of dealing with it? If the cataract is overripe, it is best to rid the eye of the débris in the aqueous chamber, because it is degenerated and pathologic material, and is, therefore, irritating and slow of absorption. If, however, the cataract is unripe, the cortical fibers left behind are not irritating and will become readily absorbed, because they are not pathologic in the above sense.

They are to be likened to extravasated blood in the aqueous chamber, which absorbs because the blood-cells and plasma were physiologic and functionating before they escaped. The principle involved is clear when the above facts are recognized.

(e) *As to time as a factor:* Too much time is usually consumed in performing the cataract operation in all its details. Cataract patients are usually old and infirm, and the consequent shock is needlessly great after an operation that consumes from thirty to forty-five minutes of time. There are two kinds of shock that must be sustained in every cataract operation—the local shock to the tissues incident to prolonged anesthesia and much instrumentation, and the nervous shock or strain incident to the full realization that so much is at stake. It is exceedingly important that this operation be over with as quickly as is compatible with a finished result. The anesthetic should be completed in eight minutes' time; the operation completed after that in from three to five minutes' time.

Every instrument that is introduced within the eye is an added risk. The fewest instruments compatible with success should be employed.

(f) *As to atropin:* The wounded eye takes to atropin badly. Whether this is due to the solution being foul, as is often the case, whether it is due to idiosyncrasy that sometimes exists when atropin solution is frequently employed, whether it blocks the drainage channel already choked with serum, loose cells, and congested iris and ciliary, or whether, indeed, the eye becomes infected from much manipulation during its use, I will not venture to state, but I am convinced, from actual experience, that atropin, except in very rare instances, is a positive detriment to the eye at any stage after extraction of cataract. It is natural that the cut eyeball in reaction should be congested. This congestion is conservative and useful in nature's process of repair. The sur-

geon ofttimes mistakes it for oncoming iritis and uses atropin. The iritis then materializes, or in some cases glaucoma.

In Conclusion.—The last word has not been uttered in the fascinating and perplexing subject of cataract surgery. We must progress in this as in other surgical matters. The old-established methods in the surgical treatment of cataract are practically those taught over fifty years ago. It is the hope of the writer that the "newer principles" mentioned will be considered along the lines of progress, and a step nearer that fond dream of the cataract surgeon: viz., a safe operation for immature cataract which any good surgeon can perform in one operation in a few minutes' time, a round central pupil, a clean black pupil, primary and rapid healing of the wound, and complete and satisfactory restoration of vision.

APPENDIX AND STATISTICAL TABLE.

In order to sustain the argument that immature cataract offers an excellent opportunity to achieve first-class results in one operation I wish to present a table of statistics, compiled from my records by my assistant, Dr. P. L. Coulter, who was instructed to record *each and every case of uncomplicated, immature, senile cataract with vision of 20/200 and better that I have operated upon since January, 1910.* It will be noted that the optic disc could be studied with the ophthalmoscope through the dilated pupil just before operation. It is a source of great satisfaction to be able to see that the fundus is normal. There were, during the period of time covered in these statistics, about twice as many more cases of immature cataract in which the vision was between 20/200 and counting fingers beyond two feet. The results from operation in this class were equally good. They were not tabulated because the argument of this paper is to support the newer principle that there is no need to wait until the cataract incapacitates the patient, and also that there is never a demand for preliminary iridectomy. In no case

was iritis or infection of any kind present. One eye was lost from choroidal hemorrhage induced by epileptic seizure that followed a smooth extraction. This was the only eye in the series that was lost. The visual results compare very favorably with any achieved after the capsulotomy operation in mature cataract; in fact, I believe are better.

The time will come when it will be taught that the most favorable form of cataract for operation is the *immature*, and operators will feel that they are jeopardizing their results by waiting for maturity.

The following table shows all cases of uncomplicated immature cataracts having vision of 20/200 and better operated on within the past five years:

CASE NO.	NAME	AGE	V. BEFORE OPERATION	OPTIC DISC SEEN	V. AFTER OPERATION
1	M. B.	60	20/70	Yes	20/35 Ja. No. 2
2	S. D.	60	20/70	Not noted	20/20 Ja. No. 1
3	A. H.	61	20/200	Yes	20/20 Ja. No. 1
4	A. F. L.	45	20/200	Yes	20/20 Ja. No. 1
5	S. W. C.	70	20/200	Yes	20/20 Ja. No. 1
6	A. F. L.	46	20/200	Yes	20/20 Ja. No. 1
7	M. C.	72	20/70	Yes	20/20 Ja. No. 1
8	M. C.	72	20/200	Yes	20/40 Ja. No. 1
9	F. B.	37	20/200	Yes	20/20 Ja. No. 1
10	S. E. M.	76	20/200	Faintly	20/30 Ja. No. 1
11	B. F. R.	68	20/200	Yes	20/20 Ja. No. 1
12	C. H. D.	49	20/200	Not noted	20/25 Ja. No. 1
13	J. R.	45	20/100	Yes	20/20 Ja. No. 1
14	C. L. W.	68	20/200	Yes	20/20 Ja. No. 1
15	A. D.	63	20/200	No	20/30 Ja. No. 2
16	R. B.	65	20/50	Yes	20/20 Ja. No. 1
17	H. S. B.	67	20/200	Yes	20/30 Ja. No. 1
18	W. Y.	77	20/200	No	20/40 Ja. No. 3
19	E. W. P.	56	20/70	Yes	20/20 Ja. No. 1
20	E. W. P.	56	20/70	Yes	20/20 Ja. No. 1
21	J. L. B.	53	20/200	Yes	20/30 Ja. No. 1
22	F. S. M.	71	20/200	Yes	Nil
23	C. L.	57	20/200	Yes	20/30 Ja. No. 2
24	L. F.	65	20/200	Yes	20/20 Ja. No. 1
25	R. J.	79	20/50+	Yes	20/30 Ja. No. 2
26	A. H.	69	20/200	No	20/30 Ja. No. 1
27	J. C. L.	70	20/200	Yes	20/20 Ja. No. 1
28	A.	70	20/50	Yes	20/20 Ja. No. 1
29	N. S.	66	20/40	Yes	20/20 Ja. No. 1
30	J. A. DeA.	74	20/70	Yes	20/20 Ja. No. 1
31	E. S. R.	75	20/70	Yes	20/20 Ja. No. 1
32	J. S.	62	20/100	Yes	20/20 Ja. No. 1
33	C. H. M.	70	20/200	Yes	20/20 Ja. No. 1
34	A. McK.	71	20/50	Yes	20/20 Ja. No. 1
35	A. V.	62	20/200	Yes	20/20 Ja. No. 1
36	G. C. V.	74	20/200	Yes	20/30 Ja. No. 1
37	E. S. R.	75	20/70	Yes	20/20 Ja. No. 1
38	J. F.	68	20/200	Yes	20/20 Ja. No. 1
39	C. H. M.	70	20/200	Yes	20/20 Ja. No. 1
40	J. T.	66	20/200	Not noted	20/20 Ja. No. 1

THE MOTAIS OPERATION FOR PTOSIS.

FRANCIS W. SHINE, M.D.,
New York.

This operation for the relief of ptosis, devised by the distinguished French surgeon and published in 1897, is to my mind a most ingenious and excellent procedure. Why it has not gained a greater popularity among the profession in America is hard to understand, unless it is due to the extreme patience and care with which it must be performed in order to secure favorable results. Although the operation as usually described may seem simple, one discovers, on undertaking a practical application of it, that it is so in theory only, for undoubtedly, of all the methods devised for the correction of this lid deformity, it is the most delicate and tedious. I think that possibly failure in the first case may have discouraged a number who have been disposed to regard the method favorably, and they have abandoned the procedure without having really given it a thorough trial. Ptosis is not of such common occurrence that an operator may be supplied with unlimited material, no matter how large a clinic he may have at his command, and yet it is only through perseverance in developing the technic that good results may be expected. My first experience with the operation, in 1907, in the clinic of the New York Eye and Ear Infirmary, led me to undertake the operation at every opportunity, yet since that time I have been able to collect only six cases for operation, in spite of the fact that the clinic is an unusually large one. I have, however, done a number on

* Candidate's thesis for membership accepted by the Committee on Theses.

the cadaver from time to time, and am thoroughly satisfied with the theory and its practical results.

If the number and variety of operations proposed for the relief of a surgical condition are evidence of their unsatisfactory status, then the various procedures and modifications proposed for ptosis are an eloquent reminder of that fact. It is my belief that the operation of Motais merits a greater number of adherents, and as the results in my six cases compare favorably with those reported by other operators, and also with the results obtained by other methods, I feel that a report of them somewhat in detail, together with a description of the operation, emphasizing what appear to be the important factors in the success of the procedure, is worthy of record.

Broadly speaking, operations for ptosis may be classed under three groups:

First, those designed by excision of the skin or tarsus or both to shorten the lid, and thus elevate the margin above the pupil, the best example being the operation of de Grandmont and Gruening's modification. The effect from this procedure, as a rule, is not great, and is only partially successful in that, while the lid may be elevated sufficiently to provide for vision, a considerable part of the deformity remains and obviously no additional motility is obtained.

Second, those in which the levator itself is resected and shortened or else tucked up by means of sutures—for example, Bowman's and Eversbusch's methods and their modifications. Although having had no experience with these procedures, I should think that this method would be limited to a very small group of cases, and chiefly valuable in traumatic cases of recent origin. It is evident that not much could be expected from operations of this kind upon long-standing paralyzed muscles or congenital ptoses.

Third, those in which the action of another muscle, superior rectus or frontalis, is substituted for the inactive

one; examples, the operations of Hess, Panas, Motais, and Parinaud.

All of these operations have met with more or less success in the hands of various surgeons, but the first two in the third class seem to have gained the greater favor, that is, those calling for the substitution of the frontalis muscle, and even they are under a great disadvantage in that the contortions brought about by the forced use of the muscle approach at times the grotesque, and if this can be avoided a great cosmetic advantage has been obtained.

Certainly the alliance between the superior rectus and the levator seems a most natural one, for, as pointed out by Motais, anatomically they lie in close relationship from origin to insertion, the levator directly above the superior rectus and the fasciæ surrounding the two muscles blending intimately. Physiologically also they are in action somewhat similar; they describe the same curve and, practically speaking, the action of one is almost always accompanied by action, to some degree at least, of the other, and lastly they are both innervated by the same nerve. These points I assume will appeal to all of us, but it is difficult to accept the enthusiastic claim of the originator that the slip of tendon from the superior rectus acquires a natural function of its own, that is, as far as any independent action is concerned. I quite agree with Shoemaker, who maintains that the effect is due to a mechanical anchoring of the lid to a fixed point on the eyeball, the location of which is very close to that from which the lid is physiologically supported. The lid, however, presents a much more natural appearance after this operation than after any other, and certainly its simultaneous elevation with the eyeball is a result more nearly approaching a normal physiologic action than can be obtained by any other method.

The operation is a limited one to this extent, that to obtain the best results the cases must be selected, though theo-

retically it is applicable to all types of ptosis with the exception of a complicating paralyzed and inactive superior rectus or a complete third nerve paralysis. The former condition has not been regarded by some as absolutely contraindicating the procedure, Shoemaker having suggested its possible efficacy even with this complication. As far as I have been able to ascertain, however, no one has had the temerity to undertake it under these conditions.

The class of cases in which it would seem to be most successful are:

1. Those of partial congenital ptosis with a fully developed elevating power of the eyeball.

2. Traumatic cases, especially those of a mild type. It is doubtful whether, in a complete traumatic ptosis with a much thickened lid, a slip from the rectus could be obtained strong enough to fulfil the desired function.

3. Cases where the vision in the eye affected is markedly deficient, or where the eye is actually amblyopic, thus lessening or avoiding the tendency to a production of diplopia.

The operation, however, may be done in any type and in ptosis of any degree, and unquestionably many cases present themselves in which a combination with other operations could be undertaken with decided advantage.

The operation is performed preferably under cocain anesthesia; besides avoiding the disagreeable effects of a general anesthetic, the patient is able to aid the operator by maintaining the proper position of the eye at each step; also the amount of apparatus in the neighborhood of the field of operation is reduced to a minimum.

A speculum is inserted, and the patient depresses the eyes well downward. A transverse incision is made in the conjunctiva over the site of the insertion of the superior rectus, about 2 cm. in length, exactly as in a resection for strabismus. The conjunctiva over the muscle is dissected well backward to the fornix. In the middle of the incision two silk sutures

are passed in the upper flap, about 4 or 5 mm. apart, the needles removed and the ends tied, leaving the sutures to act as retractors. Between these two thread retractors a vertical incision is now made in the conjunctiva in the direction of the muscle and well up into the fornix. The speculum is now removed, the lid everted, and the upper margin of the tarsus is grasped with a fixation forceps. This exposes the upper fornix, and the incision in the conjunctiva is carried on up to the upper border of the tarsus. The conjunctival flaps are dissected up, and two silk sutures are passed through the margin of the flaps near the fornix, one on each side, looped and retained to act as retractors. These thread retractors eliminate a number of instruments which would otherwise have to be used, and which would necessarily interfere with the operator. The superior rectus muscle, beginning at its insertion, is now cleared of Tenon's capsule on each side, as in a resection of the muscle. With a pair of Stephens' tenotomy scissors an incision is made at the upper border of the tarsus through the levator attachment at the point where the conjunctival incision joins it. This should be approximately the middle of the lid. The tissues on the anterior surface of the tarsus are now dissected thoroughly from the cartilage for a space sufficient to allow the subsequent introduction of the tendinous slip, the points of the scissors hugging closely the tarsus itself and extending the dissection down to the lower border of the lid. This I consider a very important step in the operation, and must be done thoroughly, leaving a fairly wide space in which the slip from the muscle may slide easily when placed in position. The next step is to obtain the slip from the middle of the superior rectus tendon. A squint hook is passed beneath the tendon, which is held away from the globe, and a fine silk suture threaded on both ends with a small curved needle is then passed from without inward through the middle of the tendon; the needle should enter

about 3 mm. behind the insertion, and care must be taken to see that a good-sized width of the tendon is included in the bight of the thread. The slip is then cut out on each side of the thread up to the actual insertion of the tendon anteriorly and back about 10 or 12 mm., and should be at least 3 mm. wide anteriorly. As the muscle widens out posteriorly for a considerable distance, it is well to fashion the slip of tendon likewise, that is, wider at its posterior end or base, especially if the slip is to extend into the belly of the muscle. If possible, when severing the anterior end of the slip, cut off the fibrous tissue attaching the tendon to the sclera; this leaves a little knob of fibrous tissue and will tend to prevent the suture from cutting through when finally attached on the anterior surface of the cartilage. This slip then having been freed, the needles are passed through the opening in the levator at the superior border of the cartilage, slipping along its anterior surface and emerging just above the cilia and about 4 mm. apart. It is difficult to pass the sharply curved needle in this manner, so that I have found it best to substitute straight needles for this step of the operation, using a curved needle on only one end of the suture when passing it through the tendon and substituting a straight one afterward. The slip is then pulled into place, the lid at the same time being pushed gently backward, and when elevated well above the cornea, the sutures are tied over a small roll of gauze. The vertical incision in the conjunctiva is then closed with three or four silk sutures, care being taken to place one or two well up in the fornix. The silk thread first used for retractors here assists greatly in passing the sutures. The transverse incision in the conjunctiva is closed in the same manner with as many sutures as may be necessary. The eye is then washed thoroughly and, the cornea being more or less exposed, boric-acid vaselin is applied freely to prevent its coming in contact with a light dressing used over the eye. Both eyes should be bandaged

for the first two days to prevent unnecessary movements of the lid. The bandage is removed about the third day, and a day or two later the sutures are removed. It is well to obtain a marked overeffect at the time of the operation, as the effect will diminish somewhat, and I have not seen any ill result or inability to close the lid after a few days following the operation in any case that I have operated, no matter how great the immediate elevation of the lid.

The following are the cases operated and results:

CASE 1.—Woman, aged twenty-nine years. Traumatic ptosis of the right lid resulting from a fall when eight years of age. Lid covers more than half of pupil. Motility of eyeball good in all directions. Uses the frontalis in efforts to elevate the lid.

May 24, 1907, operation. This was followed by considerable edema of the lid for two or three days. Suture removed on seventh day. Patient discharged with only partial correction of deformity. Seen in clinic several weeks later; result fair.

CASE 2.—Woman, aged twenty-seven years. Congenital ptosis. Both lids cover one-half of pupil. With aid of frontalis can raise the lids about 2 mm. V. R. E., = 20/40, with sph. − 2.50 D.⌓ cyl. − 2.00 ax. 45; L. E., = 20/50, with sph. − 3.00 D.⌓ cyl. − 1.50 ax. 45. Motility of both eyeballs good in all directions.

July 14, 1908, operation right eye. Marked entropion present at completion of operation, but lid raised well above the cornea. The entropion and edema of lid were pronounced for several days, so that an adhesive strip had to be used to overcome the entropion. On discharge this had been completely corrected and the result was good. No diplopia.

CASE 3.—Girl, aged seventeen years. Congenital ptosis right lid; left normal. Lid of right eye covers one-half of pupil; with the aid of frontalis could be raised 2 mm. Motility of eyeball good. V. R. E., = 20/100, with sph. −4 D; L. E., = 20/30, with cyl. +1.00 ax. 180.

July 22, 1908, operation. On fifth day lid still swollen

but drawn up well above the cornea. The result in this case was excellent. The lid, which had the usual smoothed-out appearance of congenital ptosis, assumed a normal fold closely resembling its fellow eye. The patient was much pleased with the result.

CASE 4.—Woman, aged twenty years. Partial congenital ptosis left eye, lid coming slightly down over the pupil.

June 17, 1909, operation. On the following day there was only slight elevation of the lid and some entropion. The entropion disappeared, but this operation was a complete failure. A very narrow slip was taken from the superior rectus, and apparently the suture slipped off, for in a few days there was no apparent result. This should have been a most favorable case, as the ptosis was slight and the motility good. Patient complained of slight diplopia after operation, but this disappeared.

CASE 5.—Woman, aged thirty-nine years. Ptosis left eye present since early childhood; no history as to cause. V. R. E., 20/30; L. E., 20/30.

July 8, 1914, operation. On fifth day discharged. Upper lid held well up above the cornea and excessive sagging of lid integument. This same condition was present when last seen, several months after operation, but the margin of the lid was well elevated. I was anxious to excise a strip of this redundant skin in the lid which would have greatly improved the appearance, but the patient was well satisfied with the result and refused to have anything more done.

CASE 6.—Boy, aged nine years. Congenital ptosis right eye. Lid droops so markedly that almost the whole pupil is covered. Tilts head backward, and is able, by action of frontalis, to uncover about half of pupil. V. R. E., = 20/20; L. E., = 20/30. Is highly hyperopic, right about 3 D., left about 5 D.

July 25, 1913, operation. Immediate effect pronounced, and when seen, several weeks after operation, the appearance of the lid was excellent. No diplopia. The patient was not seen again until the early part of March, 1916. At that time the appearance of the lid was quite natural, but there was a left hyperphoria of 5° and an esophoria of 10°.

The mother states that in looking up the patient does so naturally and never throws his head back, as he invariably did before operation. The boy has never complained of diplopia in spite of the hyperphoria. The cosmetic effect is excellent.

In making a choice of operation and comparing the advantages of the various procedures there are many things that commend the procedure of Motais.

First, the fact that the operation is done subconjunctivally and behind the lid, making unnecessary incisions in the skin and avoiding scars which, after other operations, often remain and are unsightly, as, for example, in the operations of Panas and Hess and their modifications. In the Panas operation there is not only a certain amount of disfigurement from the scar, but the principle of including a skin-flap underneath a denuded surface is not surgical.

Second, the effect of a properly performed and successful operation should be to give a more normal appearance to the lid by pulling it up into a natural position, with the formation of a fold in the skin of the lid resulting in complete abolition of its former smooth, flat appearance.

Third, there is little tendency to produce entropion or ectropion, although for a few days after the operation, owing to swelling of the lid, entropion may be present. This, however, disappears with the subsidence of the swelling.

Fourth, the operation is easily performed under local anesthesia.

Fifth, no other function of the lid is in the least interfered with, and even if the operation is not entirely successful it would not interfere with an operation of another character.

Hyperphoria and diplopia are possible after-effects that must be taken into consideration, although I have never had any but transient diplopia. In Case 6 I had a marked hyperphoria develop (5°) combined with an esophoria of 10°. One must consider here the possible influence of the hyper-

phoria in causing a lateral squint to develop later. This, I should think, would be more likely to occur in the case of younger patients. The hyperphoria in this case was discovered two years after the operation, which, from a cosmetic standpoint, has been a perfect success. The fact that the patient was highly hyperopic must also be taken into consideration as a possible factor in causing the esophoria, which might very well have developed independently of the operation.

After observations extending over a considerable period of the effects obtained by other operators and other methods it seems to me that the method of Motais has much to recommend it. No operation has yet been devised that will prove perfect in every respect, but from the standpoint of both visual and cosmetic improvement, the latter often the most important consideration with the patient, I have concluded that this procedure gives the most uniform and successful result.

A STUDY OF HEMORRHAGE INTO THE ANTERIOR CHAMBER SUBSEQUENT TO OPERATIONS FOR HARD CATARACT.*

JOHN M. WHEELER, M.D.,
New York City.

In ophthalmic literature it is surprising how little there is of definite value on the matter of post-operative hemorrhage in cataract cases. Hemorrhage into the anterior chamber subsequent to the different operations for hard cataract is deserving of careful study, if for no other reason than that it causes the eye to present an alarming appearance.

Allow me then to present a review of the literature upon this subject, together with a detailed report of 2123 opera-

* Candidate's thesis for membership accepted by the Committee on Theses.

tions for hard cataract performed at the New York Eye and Ear Infirmary during a period of ten years from January 1, 1899, to January 1, 1909, by many different operators, with varying amounts of experience and varying degrees of skill.

REVIEW OF LITERATURE.

Of all ophthalmic surgeons, Professor Herman Knapp has given us by far the most in the way of detailed reports on the subject of after-hemorrhage in cataract extractions. In series of 100 cases he reported in detail his operations, with results, mounting up to several hundreds. In one series of 100 successive cases of cataract extraction without iridectomy,[10] no hemorrhages into the anterior chamber were reported. In another series of 100 without iridectomy,[11] he speaks of "several cases of secondary hemorrhage into the anterior chamber, all traumatic, all without consequence." In another series of 100 cases of extraction by the peripheric-linear method,[12] he reports 6 after-hemorrhages: 5 occasioned no harm; the sixth case developed an iritis, and a few months after extraction sympathetic inflammation was set up, occasioned, Knapp thought, by the hemorrhage. This case he considered "gave proof that after-hemorrhage, although having generally no bad consequence whatever, may in exceptional cases lead to severe iritis and even cause sympathetic trouble in the other eye." In a series of 200 cases, performed according to the von Graefe method,[13] there were 6 after-hemorrhages into the anterior chamber. In yet another series of 100 cases,[14] performed according to the method of von Graefe, there were 8 cases of after-bleeding with no bad results. In another series of 100,[15] after-hemorrhage was noticed "repeatedly" and without consequence. In another series,[16] which Knapp terms his eighth, there were 3 after-hemorrhage cases. He[17] also issued a report in 1897 on 400 unselected cases with only 1 post-operative hemorrhage into the anterior chamber. He said that he could

positively ascribe hemorrhage to injury in a large number of after-hemorrhages, but he was satisfied that some cases had another cause for bleeding, although he could not tell what it was.

Hasket Derby[5] in 1873 gave an analysis of 64 cases of cataract extraction operated on at the Massachusetts Charitable Eye and Ear Infirmary in 1872 by the surgeons of the Infirmary, and reported 4 cases of hemorrhage into the anterior chamber, 3 of which occurred from accidents. In 1881 he reported 100 cases of cataract extraction,[6] performed by himself according to the method of von Graefe, and mentioned only 1 post-operative hemorrhage which occurred five days after operation.

In 1874 C. R. Agnew[1] submitted a detailed report of extraction by different methods without hemorrhage into the anterior chamber.

Kipp,[8] in 1880, published a small series of cataract extractions performed under "unfavorable hygienic conditions." Out of 10 cases, only 1 suffered from hemorrhage into the anterior chamber. This was a patient who had an attack of acute inflammatory rheumatism. Several hemorrhages occurred without producing noticeable reaction.

Franke,[7] in 1881, cited 1 case of hemorrhage into the anterior chamber in a series of 97 cases of extraction.

Out of the 261 cases of cataract extraction performed by Dr. George Strawbridge,[22] and reported in 1885, 1.92 per cent. had post-operative hemorrhage. Of these hemorrhages, 3 occurred four days after operation; 1 occurred five days after; 1 case had hemorrhages twenty-four hours and forty-eight hours after operation; and 1 case, one month after operation and two weeks later.

In 1890 A. Neve[20] published an analysis of 100 cases of cataract extraction without iridectomy, reporting 4 hemorrhages into the anterior chamber.

Arnold,[2] in 1893, reported 18 hemorrhages (6.59 per cent.)

into the anterior chamber in 273 cases of combined extraction.

In 1895 C. Devereux Marshall[19] published a detailed report of 1519 cases operated on at the Royal London Ophthalmic Hospital from 1889 to 1893. Of these cases 19, or 1.25 per cent., had hyphemia following operation. An interesting and rather remarkable feature of this report is that, although 7 of these cases had albumen and 30 of them had sugar in the urine, not one of the nephritic or diabetic patients is recorded as having hyphemia.

Schreiber[21] reported, in 1886, 103 cases of cataract extraction (10 with iridectomy), and 9 of these cases had hemorrhage into the anterior chamber.

Köllner,[18] in 1907, reported 1284 cases of extraction with iridectomy, with 67 instances of hemorrhage into the anterior chamber, or 5.22 per cent. He mentions, as causes, displacement of bandage, touching with hands, violent moving, coughing, pressure, etc.

In considering the subject of cataract in diabetics, W. Uhthoff, of Breslau,[23] in 1908, reports a series of 115 cataract extractions in diabetics with favorable results. He considers the results as good in diabetics as in non-diabetics. In his series of diabetic cases post-operative hemorrhage into the anterior chamber occurred in 8.9 per cent.

In the discussion of post-operative complications of cataract extraction at the 1914 meeting of the Ophthalmological Society of the United Kingdom Mr. Claude Worth[24] spoke of hemorrhage into the anterior chamber. In the cases which he had seen, hemorrhage occurred almost exactly one hundred and twenty hours after operation, and he thought that this accident was peculiar to the cataract extraction with a conjunctival flap. He had never seen it follow operations with corneal incision. He thought that the hemorrhage had come from the vessels, which Mr. Collins had demonstrated, growing down from the conjunctival flap.

Mr. Worth's observations are interesting, but at variance with those of other oculists in the exactness of the time of hemorrhage, and his opinion in regard to the source of bleeding is not in accord with that held by most observers, who believe hemorrhage comes from the iris.

It is exceedingly interesting to note that in 100 successive extractions of cataract in the capsule reported by Arnold Knapp[9] in January, 1915, there was only 1 case of hyphemia.

H. Becker[4] and Benjamin J. Baldwin[3] each reported a case of hemorrhage from the iris following cataract operation, so severe that life was threatened, but in each case the hemorrhage immediately followed iridectomy and continued until the condition of the patient was considered serious. These cannot be classified as after-hemorrhages.

The 2123 operations which I have analyzed include all the preliminary iridectomies, extractions with iridectomy, extractions after iridectomy, simple extractions, and extractions in capsule which were performed at the New York Eye and Ear Infirmary by the surgeons, assistants, and members of the house staff during the period from January 1, 1899, to January 1, 1909.

This is an interesting period for this study, as it is one of transition in the method of operation. During this period the number of simple extractions decreased, while the number of extractions accompanied by iridectomy or following iridectomy increased. Of these 2123 operations, 459, or 21.6 per cent., were preliminary iridectomies; 778, or 36.6 per cent., were extractions with iridectomy; 352, or 16 per cent., were extractions after iridectomy; 528, or 25 per cent., were simple extractions, and 6, or 0.28 per cent., were extractions in capsule.

Careful records of the post-operative conditions of these patients were kept by the house surgeons. Unfortunately, ultimate vision was given in so small a percentage of the cases that I have not attempted to tabulate the visual re-

sults. Probably, also, an allowance should be made for a slight error in the number of hemorrhages which are recorded, as it would be possible for a few incidents in the healing process to escape the observation of the house officers. The number of recorded hemorrhages would be too small, rather than too large, in all probability. The figures, however, are interesting and approximately correct.

In all there were post-operative hemorrhages into the anterior chamber in 97 eyes. These occurred from one day to twenty-eight days after operation. In these cases 76 had 1 hemorrhage, 15 had 2 hemorrhages, 3 had 3 hemorrhages, 1 had 4 hemorrhages, and 2 had repeated hemorrhages without record of the number.

The following table gives the details as to the number of hemorrhages occurring following the different methods of operation.

TOTAL NUMBER OF HEMORRHAGES OCCURRING AFTER OPERATION.

Number of Hemorrhages	Preliminary Iridectomy	Extraction with Iridectomy	Extraction after Iridectomy	Simple Extraction	Extraction in Capsule	Total
1	8	41	9	17	1	76
2	1	10	1	3	0	15
3	1	0	1	1	0	3
4	0	1	0	0	0	1
Repeated	0	2	0	0	0	2
Total	10	54	11	21	1	97

4.57 per cent. of the entire number of cases had hemorrhage.

2.17 per cent. of the preliminary iridectomies had hemorrhage.

6.94 per cent. of the extractions with iridectomy had hemorrhage.

3.12 per cent. of the extractions after iridectomy had hemorrhage.

3.97 per cent. of the simple extraction cases had hemorrhage.

16.66 per cent. of the extraction in capsule cases had hemorrhage.

The number of extractions in capsule is so small that this comparison is too unfair to this method of operation to be considered.

INFLUENCE OF AGE.

For the purpose of comparison I have divided the 2123 cases into 6 age groups of ten years each, and have found the percentage of post-operative hemorrhage cases in each group as below:

AGE	NUMBER OF CASES	NUMBER OF HEMORRHAGE CASES	PERCENTAGES
30–40	71	2	2.82 per cent.
40–50	251	19	7.57 " "
50–60	591	23	3.89 " "
60–70	755	35	4.64 " "
70–80	395	15	3.8 " "
80–90	56	1	1.79 " "

In 4 cases of the entire number no age was given. Of these, 2 cases had after-hemorrhage.

Thus it is seen that, in this series of cases, the surprising fact is brought out that more hemorrhages proportionally occurred in patients from forty to fifty years of age than in those over fifty years, when it might be expected that sclerosis would be more advanced and the vessels more liable to leakage. It is possible that this may be accounted for by greater activity and greater liability to injury on the part of middle-aged patients, and this may more than counterbalance the

activity occasioned by dementia and irresponsibility on the part of older patients.

The following additional tables give detailed information as to the number of hemorrhages and the time of their occurrence in patients of different ages, following the different kinds of operation in these cataract cases.

CASES HAVING ONE AFTER-HEMORRHAGE.

Operation	Age	Number of Days After Operation
Preliminary iridectomy	52	4
	60	3
	60	4
	63	2
	64	3
	65	3
	70	3
	78	6
Extraction with iridectomy	40	6
	43	3
	44	17
	45	5
	45	2
	48	4
	48	2
	48	No date
	50	4
	50	1
	50	4
	52	5
	52	28
	54	3
	55	4
	57	6
	57	18
	59	4
	59	4
	60	5
	60	3
	60	16
	60	5
	61	2
	62	6
	64	5
	64	6
	65	2
	65	2
	67	7
	68	3
Extraction with iridectomy (*Continued*)	68	3
	69	6
	70	3
	70	13
	70	5
	71	10
	72	6
	74	4
	75	4
	76	4
	76	5
	79	4
	No age	7
Extraction after iridectomy	41	3
	44	5
	50	5
	62	10
	74	4
	80	5
Simple extraction	35	5
	36	3
	40	7
	42	9
	45	8
	50	3
	55	6
	55	4
	58	5
	58	4
	61	16
	66	3
	66	6
	67	5
	68	6
	68	11
	No age	3
Extraction in capsule	54	3

CASES HAVING MORE THAN ONE HEMORRHAGE.

OPERATION	AGE	NUMBER OF HEMORRHAGES	NUMBER OF DAYS AFTER OPERATION
Preliminary iridectomy	49	2	4, 7
	67	3	4, 6, 7
Extraction with iridectomy	42	2	4, 7
	47	2	3, 11
	47	2	6, 11
	47	2	5, 14
	54	2	6, 12
	60	4	3, 10, 12, 15
	60	2	10, 20
	65	2	5, 9
	65	2	3, 8
	65	2	2, 4
	66	2	3, 9
	66	Repeated	
	72	"	
Extraction after iridectomy	47	3	5, 8, 9
	55	2	7, 16
Simple extraction	54	3	1, 4, 6
	55	2	2, 4
	60	2	3, 9
	77	2	6, 12

INFLUENCE OF DIABETES.

In this series of 2123 cases 38 were recorded as having sugar in the urine. This is 1.78 per cent. of the entire number. Of these 38 diabetic cases, 11, or 28.94 per cent., had hemorrhages. In so far as the percentage of the amounts of sugar were given, the figures prove interesting.

Twelve cases had less than 2 per cent. of sugar in the urine, and of these 4 had hemorrhage.

Eight cases had more than 2 per cent., and of these 1 had hemorrhage.

Eighteen cases had sugar in the urine, but the amount was not given, and of these 6 had hemorrhage.

Comparison of the percentage of the total number of cases having hemorrhage (4.5 per cent.) with the percentage of diabetic cases having hemorrhage (28.94 per cent.) shows an overwhelming tendency to after-bleeding into the anterior chamber where sugar is present in the urine.

Judging from these figures, it would appear that a large

quantity of sugar in the urine would not indicate a greater tendency to postoperative hemorrhage than a small amount; but the number of diabetic cases is too small for a valuable comparison.

INFLUENCE OF ALBUMINURIA.

The records show the presence of albumin in 31 cases, or 1.46 per cent., of the whole series. A quantitative analysis was recorded in so few instances that no attempt will be made to analyze the influence of nephritis in so far as the percentage of albumin present in each case is concerned. In these 31 albumin cases there were 4 hemorrhages. In other words, hemorrhage occurred in 12.9 per cent. of the cases. So it will be seen that these albuminuric patients are less prone to after-hemorrhage than are diabetic patients, but more liable to hemorrhage than those having neither albumin nor sugar.

I wish that it might be possible to make an accurate study of the comparative visual results in these cases, but the incompleteness of the records renders any figures that might be given almost valueless. However, from the study of the literature and such results as are recorded in this series one must gain the impression that this apparently alarming incident in the healing process following operation for hard cataract has very little influence on the final results. This seems to be generally agreed upon.

CONCLUSIONS.

From the analysis of this series of 2123 operations, the following conclusions may be drawn:

1. Hemorrhage into the anterior chamber following operation for hard cataract occurs in about 4.57 per cent. of the cases.

2. Hemorrhages are more liable to occur in middle-aged patients than in either the young or the old.

3. It may occur from one to twenty-eight days after operation, and may take place once or a number of times in the same eye.

4. Diabetes especially predisposes to post-operative hemorrhage, and the amount of sugar appears to have little influence on the likelihood of this event.

5. Albuminuria predisposes to hemorrhage, but less markedly than glycosuria.

6. In the different methods of operation there is greatest liability to post-operative hemorrhage in "extraction with iridectomy." There is less liability to hemorrhage in "simple extraction." There is still less in "extraction after iridectomy," and least of all in "preliminary iridectomy."

REFERENCES.

1. Agnew, C. R.: Trans. Amer. Ophth. Soc., 1874, ii, p. 251.
2. Arnold: Arch. f. Augenheilk., xxv, S. 41.
3. Baldwin, Benj. J.: Arch. Ophth., 1889, xviii, p. 328.
4. Becker, H.: Arch. Ophth., 1905, xxxiv, p. 670.
5. Derby, Hasket: Arch. Ophth., 1873, iii, p. 74.
6. Derby, Hasket: Trans. Amer. Ophth. Soc., 1881, iii, p. 195.
7. Franke: Arch. Ophth., 1881, x, p. 121.
8. Kipp: Arch. Ophth., 1880, ix, p. 171.
9. Knapp, Arnold: Arch. Ophth., 1915, xliv, p. 1.
10. Knapp, H.: Arch. Ophth., 1889, xviii, p. 1.
11. Knapp, H.: Arch. Ophth., 1890, xix, p. 280.
12. Knapp, H.: Arch. Ophth., 1869, i, p. 103.
13. Knapp, H.: Arch. Ophth., 1877, vi, p. 3.
14. Knapp, H.: Arch. Ophth., 1879, viii, p. 200.
15. Knapp, H.: Arch. Ophth., 1881, x, p. 295.
16. Knapp, H.: Arch. Ophth., 1883, xii, p. 69.
17. Knapp, H.: Jahres. über Ophth., 1897, p. 95.
18. Köllner: Zeitschr. f. Augenheilk., xv, S. 507.
19. Marshall, C. Devereux: Royal London Ophth. Hosp. Rep., 1895, xiv, p. 56.
20. Neve, A.: Lancet, ii, p. 869.
21. Schreiber: Jahres. über Ophth., 1896, p. 98.
22. Strawbridge, Geo.: Trans. Amer. Ophth. Soc., 1885, iv, p. 298.
23. Uhthoff, W.: Arch. Ophth., 1909, xxxviii, p. 49.
24. Worth, Claude: Trans. Ophth. Soc. U. K., xxiv, p. 67.

THE ETIOLOGY OF RETINITIS CIRCINATA.*

EUGENE M. BLAKE, M.D.,
New Haven, Conn.

The frequency with which retinal hemorrhages are found in cases of retinitis circinata has caused considerable discussion as to whether or not the latter is in any way dependent upon the former. A case of retinitis circinata seen by the writer during the past year was so suggestive of a development of this condition from an extensive hemorrhagic process into the retina that it will, possibly, bear relation.

Mrs. H., widow, American by birth, aged seventy-nine years. Father died at sixty-four of pneumonia; mother at fifty-seven of tuberculosis. Neither had had any eye troubles. Her own health has always been excellent, and she now lives alone, at the age of eighty, in a small village and does her own housework. Can remember having had no serious illnesses during her lifetime, and never had any eye trouble except hypermetropia and presbyopia until now, and attributes her present affliction to "high living and no exercise" while on a visit to her son, who is living and in good health, aged fifty-four years.

Patient was first seen by the writer on March 2, 1915. Aside from sclerotic changes in the retinal vessels, rather to be expected than otherwise, there were no gross pathologic changes. With sph. + 2.75 D. in each eye V. = 20/25; with both, 20/20−. With sph. + 2.50 D. added J. 1. Scattered about both fundi were numerous minute, pale white dots of pigment absorption or degeneration. This was considered to be due to her angiosclerosis, with resulting lack of nutrition of the retinal and choroidal elements. The patient was not seen again until June 2d, when she came because of a

* Candidate's thesis for membership accepted by the Committee on Theses.

sudden blur over the left eye of two weeks' duration. The ophthalmoscope showed an extensive hemorrhage into the macular region and extending far beyond. Many small hemorrhages were scattered about beyond the edge of the large clot. She was sent to her family physician for general examination and treatment. He reported two days later that the systolic blood-pressure was 200 mm.; urine, sp. gr. 1.010, contained no sugar or albumin, but some casts. His diagnosis was chronic interstitial nephritis. The patient had been a heavy eater, especially of meats, and was, of course, placed on a restricted diet. Small doses of potassium iodid and cathartics were ordered.

I did not see the patient again until November 1st. There was no change in the appearance of the right eye, but to my surprise the ophthalmoscope showed a fairly typical retinitis circinata in the left eye. There was a nearly complete wreath surrounding the macula, broken above in the upper temporal quadrant, and below in a corresponding place. The macula was of a yellowish cast, and V. = fingers at five feet. Owing to lack of time, as the patient was leaving for home in a distant part of the State, I made only a rough sketch of the fundus. The plaques composing the girdle were white, slightly elevated above the level of the surrounding retina, and somewhat thicker near the papilla. Just at the lower edge of the circinate wreath were several small hemorrhages.

I had no further opportunity to study this case until March 2, 1916, when I made a trip to her home for that purpose, and found the condition of the left eye as follows: The papilla was of a yellowish-pink hue and not clearly defined, a typical nerve-head of arteriosclerosis. The vessels were tortuous, the arteries small and whiter than normal, and the veins large, dark, and tortuous. Beginning close to the nerve, and extending in the form of an oval, was a dirty white wreath surrounding the macula. The latter looked edematous and more yellow than normal. The wreath looked as if composed of many smaller white spots which had coalesced. The spots began close to the disc, arched upward and out, and, after being interrupted for a short distance at the upper temporal part, curved downward again. The lower half of the oval was lacking at the extreme

temporal part and was thicker near the nerve. Just below the nerve were two small hemorrhages, about two-thirds the size of the disc, and in the lowest part of the fundus several minute hemorrhagic foci. The inferior temporal branch of the vena centralis curved in a very tortuous manner just below the inner part of the oval. At the extreme temporal part of the wreath were some slight pigment deposits, but elsewhere pigment accumulation was not seen. The exudate, if such it be, composing the circinate wreath was very little elevated and appeared less thick than when seen four months previously. On the nasal side of the disc was a small crystal of cholesterin.

The pathologic process, now called retinitis circinata, was first described by Hutchinson* under the title of "Symmetrical, Central Chorioido-retinal Disease Occurring in Senile Persons."

There are at least six different etiologic explanations offered to account for the picture of retinitis circinata:

(1) In 1893 Fuchs gave the name now generally applied, and described 12 cases, 8 of which were typical and 4 somewhat atypical. He gave it as his opinion that the white plaques were the result of an albuminous transudate into the deeper layers of the retina, a process similar to that which occurs in albuminuric retinitis.

(2) Weeks, who has seen 9 cases, would assign the cause to changes in the blood-vessels. He thinks that "the white patches are due to coagulation of fibrinous exudates which occur in the retina beneath the nerve-fiber layer as a result of changes in the small vessels of the affected zone." Fatty degeneration may occur in the plaques, adding to their brilliancy. This is practically the view held by Nuel, who would limit the site of the exudates to the so-called layer of Henle. He gives this anatomic explanation for the presence of the circle of plaques only on the temporal side of the papilla, as Henle's layer is here much thicker than on the nasal side.

* Royal London Ophthalmic Hospital Reports, 1876.

Weeks further says that the spots do not, in all probability, originate from hemorrhages, but hemorrhage and fibrinous exudate are probably dependent upon the same condition, namely, degeneration of the walls of blood-vessels.

(3) de Wecker believes the white spots are the result of fatty degeneration of previous hemorrhages. He bases this claim upon long-continued observation of the changes in the hemorrhages and in the white patches and the production of the latter, in many cases, from the former.

(4) The theory of Marcus Gunn finds some supporters. The explanation which he offers is that the white areas are simply one of the manifestations of long-continued edema, this marking out the periphery of the affected area, the size of the ring varying according to the amount of the previous affection. This view has the support of de Schweinitz, who suggests that puckering changes occur at the edge of the edematous area, where it comes into contact with the normal retina, and that later the folds assume the picture we find in retinitis circinata. A readjustment of the folds to the normal would, therefore, explain the disappearance of the white ring, which he has observed twice. Inasmuch as several cases have been followed for considerable periods, and hemorrhages never observed at any time, de Schweinitz thinks it too much to suppose that hemorrhages must always occur.

(5) Goldzieher ascribes the cause to vascular changes, occurring especially in the group of vessels which supply the macular region. He supposed these vessels to be obliterated and the parts supplied by them to degenerate. This is really what Oeller means when he says that the disease is one affecting the smallest macular arteries.

(6) This explanation is the only one which has been submitted to a microscopic examination and is offered by Ammann. His patient, a man of sixty-six, had been under observation for nine years and died during an ophthalmoscopic

examination. When first seen, several small white patches were found in the macular region and two small hemorrhages between the macula and papilla. The addition of new patches of white was seen to occur from time to time. These often coalesced to form larger plaques.

There has been some question as to whether Ammann's case is a true example of retinitis circinata, but the changes are so closely those found in this condition that it is, assuredly, of great interest.

As this case alone offers any evidence adduced from histologic examination, it will be set down in some detail. A conspicuous feature of the microscopic slide was the great hypertrophy of the supporting fibers of Müller. They were thickened and widened into wedges, base forward, in the nerve-fiber layer. Vacuolation in the several layers of the retina was found, with normal retina between the vacuoles. In the inner granular layer, beginning close to the nerve, were a number of closely placed long clots at right angles to the retinal surface. The clots presented sometimes a homogeneous appearance and sometimes granular. They stained poorly with most stains, but very well with aniline colors. Close to the clots, or masses of white, extravasations of blood and hyaline plates were seen, often broken into by a large vacuole filled with fine fibrinous exudate. These changes were found only in the inner granular layer. The hemorrhages and hyaline plates were not always two distinct forms, but were, in places, mixed. In one place a cluster of red blood-corpuscles would be found surrounded by a ring of homogeneous substance, which is colored red by eosin. In other places were hemorrhages, with one or more white clots in the center. The white plaques also varied in structure, some being perfectly homogeneous and others apparently stratified. Ammann considers these latter forms to be of great interest, as they allow an insight into the nature of the formations, for, besides plates with indistinctly outlined

granules, there are some with sharply contoured ones, and the better defined these are, the more closely they resemble red blood-corpuscles.

Another evidence of the transformation of hemorrhage to white mass is shown in a section deeply stained with van Gieson's solution. This shows the supporting structure of a pale pink color, the red blood-corpuscles yellow, and the clots intensely red. The more hyaline spots were intensely red and shining, the more granular of an orange tint, and between these two were all gradations of color.

These facts lead Ammann to conclude that the hyaline plaques are the end-products of the disintegration of red blood-corpuscles. None of the color reactions was conclusive as to which of the albuminoid bodies (hyaline, colloid, etc.) was present.

Sections treated with osmic acid showed large mononuclear cells giving a typical fat reaction. These are the well-known "fat-granule cells," concerned with the removal of red cells. The material stored up in these cells is either fat or has undergone fatty degeneration. These fat-cells were found only in places where the white spots had been seen by the ophthalmoscope. The fat-cells probably arise from the endothelium of the lymph-spaces surrounding the blood-vessels. They could not be white blood-corpuscles, as the polynuclears are the phagocytes and these cells are mononuclear. Several blood-vessels were found filled with these fat-cells.

Thickening of the intergranular layer gave the retina a size four times its normal. The outer granular layer and the layer of rods and cones were missing in the affected regions. Transition from hemorrhage to white clot and, further on, to fatty degeneration was frequently seen.

The conclusion of Ammann's most interesting case, given by him in italics, is worth recording verbatim: "Our observations show positively that the white spots are brought about only and alone by fat-cell clusters, and, further, that these

appeared where formerly hemorrhages had been found, but only at a time when nothing more of the hemorrhage was to be seen at that point.

"The hyaline clots are the result of the disintegration of red blood-corpuscles, and not, as has been thought until now, the result of exudation of albuminous fluid. Ophthalmoscopically they are either not visible at all, or cause a dirty red discoloration of the retina affected. The fat-cells have the significance of phagocytes, and probably originate from endothelial cells."

The suggested origin of the white spots from hemorrhages needs only the clinching evidence of ophthalmoscopic observation, for up to now no one has witnessed this transformation. (The case observed and here reported by the present writer seems significant, as the retinitis circinata was positively not present before the hemorrhages appeared, but was found after the latter had cleared. The actual transformation was not, however, observed.)

de Wecker, by the help of Masselon's drawings, was able to prove that, at points where hemorrhages were first seen, there were found later white plaques.

Many interesting features are disclosed by a study of the cases reported in the literature. In a patient observed by Fridenberg there was a development of new-formed vessels in the retina. He is the only writer to mention this condition. At times the disease is chronic and remains unchanged for long periods; other cases are slowly but surely progressive, and, rarely, the lesions partially or wholly disappear. In one of de Schweinitz's cases the lesions (not perfectly typical) entirely disappeared in four months, and in one case studied by Weeks the white plaques cleared away in one year. Other observers have reported improvement in the fundus condition.

On the other hand, de Wecker watched one case for over twenty years and there was no tendency toward clearing.

Cholesterin crystals were found in several cases. One patient (de Schweinitz) was the subject of adiposis dolorosa. Weeks has seen minute branches of the retinal arteries pass directly into the white patches.

Most observers place the frequency of retinitis circinata at one case in 10,000 eye patients. I cannot but think that the disease is of greater frequency than this, and that cases are either not reported or not recognized. I have personally seen four cases.

Fox states that the lesions are sometimes found on the nasal side of the papilla, but I can find no report of such a case. If this is correct, it would cast doubt upon the validity of Nuel's observation that the changes are confined to Henle's layer.

In Van Geuns' case (1908) there was a small peripheral detachment of the retina, which later became reattached. The lower half of the ring disappeared and the upper became yellowish. At the end of four months the whole ring had cleared up.

The only patient who was mentioned as being syphilitic was one of Bruner's cases, and he did not think the eye disease was in any way related to the specific trouble. In only a few of the cases has there been any nephritis, and this latter can also be excluded from the etiology.

The youngest patient was twelve, reported by Weltert. My own case, now eighty, is the oldest. Females are affected in the proportion of two to one of males. The disease is not a true inflammation, but essentially a degenerative process.

As to treatment, most authors agree that there is little to be done. de Schweinitz, however, feels that until we know more definitely the cause we should give absorptive and eliminant remedies, such as sodium iodid and mercury. In this Weltert agrees with him, believing that the choroid plays an important part in the process, and that medicines which

induce absorption exercise a favorable influence upon the vision, a fact supported by the long retention of normal vision in some cases.

In conclusion a word must be said as to the relation of hemorrhage to the white patch. It seems to me that my case is unique in that the eye was seen before either hemorrhage or exudate had appeared, and that the retinitis circinata followed so positively in the wake of the hemorrhages. Ammann suggests that hemorrhages are always present, but may be so minute that they are not visible with the ophthalmoscope, and offers as proof the fact that many hemorrhages were demonstrable under the microscope where none was seen by ophthalmoscopy. Beard says that hemorrhages are few in the beginning, but later become a constant feature of the ophthalmoscopic picture. Inasmuch as many competent observers have watched retinitis circinata grow and progress and have never seen a hemorrhage at any time, Ammann's view is hardly likely to find general acceptance. On the other hand, the very common association of hemorrhages with retinitis circinata cannot help but make one ponder as to the relationship between them. Parsons feels that the evidence is not sufficient to be positive that all the changes are dependent upon previous hemorrhage. He says that "it is probable that albuminous coagula play a part, for the resemblance to the white spots of albuminuric retinitis cannot be fortuitous, and this is essentially due to edema and not to hemorrhage."

J. W. Fischer, writing in 1913, relates four cases in support of the hemorrhagic origin of retinitis circinata. In one case in particular he found hemorrhages on the one hand, and the characteristic retinal changes of a circinate type on the other, in which the transition seemed unbroken.

My own feeling is that retinitis circinata usually develops from a previous hemorrhagic process in the retina, but I do not feel that the evidence is sufficiently strong to assert

that retinitis circinata may not, at times, arise in cases in which there are no demonstrable hemorrhages.

LITERATURE.

The literature of retinitis circinata has been brought up to date of 1913 by Heinricy and Harms, and is to be found in Graefe's Archives für Ophthalmologie, v. 36, p. 514. It includes in all 40 unilateral cases and 23 bilateral.

The additions since Heinricy and Harms published a bibliography are as follows:

Batchez, E.: Klin. Monatsb. f. Augenhk., 1913, n. F., xvi, 232–238.
Doyne and Stephenson: Trans. Ophthal. Soc. Unit. King., 1913–4, xxiv, 91.
Fischer, E. C.: Trans. Ophthal. Soc. Unit. King., 1913.
Ischreyt: Klin. Monatsb. f. Augenhk., 1914, n. F., xvii, 681.

THE ELECTROMAGNET AND THE MAGNET OPERATION IN OPHTHALMIC SURGERY.*

JOHN H. OHLY, M.D.,
Brooklyn, N. Y.

The most ancient writings record the observation that iron ore, which is rich in oxid, often possessed the power to attract small particles of iron and steel.

Homer, Plato, and Aristotle also record the fact that a piece of steel could be magnetized by iron ore with magnetic properties, and would retain this power to a more or less degree. This made possible the magnetic needle, claimed to have been used by the Chinese as a compass in 1000 B. C. The phenomenon is mentioned by Greek and Roman writers of the first century, that a piece of iron, when brought in contact with a magnet, or held near one, becomes inductively magnetized and acquires the power of lifting iron. When the iron ore is soft, such attractive power is rapidly lost if the ore is removed from the magnetic influence. If it is hard, such as steel, some of the induced magnetism is retained, and the metal becomes an artificial magnet.

* Candidate's thesis for membership accepted by the Committee on Theses.

Until 1820 all magnets were made by bringing iron ore of some form in contact with magnets or magnetic ore bodies, steel being the form of iron ore most generally used.

Every magnet has two poles, a north pole and a south pole. The magnetic force or magnetic current arranges itself in radiating lines, known as Faraday's lines, about the poles, and it was found that the poles of one magnet would attract the opposite poles of another magnet; that is to say, that the north and south poles of one magnet would attract respectively the south and north poles of another magnet. Thus, when a magnet is held near to or brought in contact with a piece or particle of iron or steel, the latter becomes magnetized and itself becomes a magnet, the north pole of the magnet attracting the south pole of the magnetized piece of steel and repelling its north pole; and in accord with this fact the law of magnetic force between poles was established by C. A. Coulomb[1] in 1785, namely, "The force of attraction or repulsion exerted between two magnetic poles varies inversely as the square of the distance between them."

Hans C. Oersted,[2] of Copenhagen, in 1820, was the first to observe that magnetism was induced by an electric current. By uniting the poles of a voltaic pile with a platinum wire, he was able to produce a condition which affected the magnetic needle. He also observed that when there was no current passing through the wire the needle was not deflected. Later in the same year A. M. Ampere[3] (1820) demonstrated that the voltaic pile itself would affect a magnetic needle as well as the wire uniting the poles, and he showed that the effects in both cases were consistent with the theory that "electric current was a circulation around a circuit, and equivalent in magnetic effect to a short magnet with its axis placed at right angles to the plane of the circuit." Upon this he advanced the hypothesis that the magnetization of iron was due to the molecular electric currents. Arago[4]

(1820) established that wire wound in a helix carrying an electric current would magnetize a steel needle placed in the interior. An electric current free to move tends to revolve around a magnetic point was demonstrated by Faraday's well-known experiment in 1821.

William Sturgeon[5] constructed an electromagnet in 1825. It was a horseshoe-shaped bar of soft iron, coated with varnish, on which was wrapped a spiral of bare copper wire, the turns of the wire not touching each other. When a voltaic current was passed through the wire, the iron became a strong magnet, but when no current passed, the soft iron lost nearly all of its magnetism.

Joseph Henry,[6] in 1831, described a method of winding electromagnets by covering the copper wire with silk, thereby insulating it, and allowing him to make many more turns of the copper wire around the horseshoe-shaped core. This increase in the number of turns of copper wire around the core made the magnet much more powerful. About this time[7] Henry constructed a magnet along these principles for Yale College, which had a lifting capacity of some 3000 pounds, although a comparatively small battery was used. This event was followed rapidly by the construction of various-shaped magnets and variations in winding, all based upon the same general principle. The strength of the electromagnet depends upon the size of the core, the diameter of the wire used for wrapping, the number of windings, and the strength of the current (amperes of electricity) flowing through the coil, *i. e.*, ampere turns.

The molecular theory of magnetism advanced by W. E. Weber[8] is briefly as follows: That the molecules of a ferromagnetic metal are small permanent magnets, the axes of which under ordinary conditions are turned indifferently in every direction, so that no magnetic polarity is exhibited by the metal as a whole. A magnetic force acting upon the metal tends to turn the axis of these little magnets in one

direction, and thus the entire piece acquires the properties of a magnet. Furthermore, Weber assumed that each molecule was acted upon by a force tending to preserve its original position, for otherwise the smallest magnetizing force would suffice to develop the highest degree of magnetism, and that this force of the returning to its natural position accounted for the loss of magnetic action.

The residual magnetism was explained by Maxwell,[9] which was, if the deflection of the axis of the molecules exceeded a certain angle, the axis would not return to its original position when the deflecting force was removed, but would retain a new permanent position.

J. A. Ewing[10] placed upon a board within a magnetizing coil many small compass needles pivoted on sharp points a short distance apart, and demonstrated that the forces by which the molecular magnets are constrained are simply those due to their own mutual attractions and repulsions.

In 1897–98 J. J. Thomson[11] advanced the electron theory. Electrons are the ultra-atomic particles carrying negative charges of electricity. They are assumed to be 1/2000 the size of an atom of hydrogen, are present in all matter, and are supposed to move freely within the atom and molecule by electric force. The action of electric force upon these electrons in ferromagnetic matter is now believed to produce the magnetic properties.

The magnet was first used in medicine nearly two thousand years ago.[12] Sucruta Ayuveda mentions the use of the magnetic ore to extract points of broken steel arrows (Hirschberg).[13]

Brunschwyek,[14] in 1462, advised the extraction of iron splinters from the eye with a magnet.

William Fabry,[15] in Hilden, used a magnet for the removal of a superficial foreign body (iron) from the cornea.

Morgagni,[16] in 1779, failed to remove a foreign body from a corneal abscess.

Milhes,[17] in 1745, reports removing one successfully.

Himley,[18] in his text-book, mentions the magnet and cites the case of Dr. Meyer, in Minden, removing with a thirty-pound magnet a foreign body which had penetrated the sclera and lodged under the iris.

Dixon[19] (1858) reports the successful extraction of a steel scissor-blade from the vitreous, drawing the same in such a position that it was removed through the scleral incision by forceps.

McKeown,[20] in 1874, extracted a piece of steel from the vitreous through the wound of entrance, by entering the magnet tip into the wound and vitreous. He used a small magnetized steel rod. The result was good, with useful vision.

To Hirschberg[21] is generally credited the early development of the magnet operation. In 1875 he constructed a useful electromagnet which consisted of a closely wrapped coil of fine insulated copper wire about a soft iron bar, one end being shaped into a point. It was used with a zinc carbon battery. This magnet was not strong enough to extract a foreign body from the eye unless the point of it was brought in close proximity or touched the same. This same type magnet, somewhat modified and improved, is still extensively used. Later other magnets were developed, and in 1881 Snell[22] constructed a magnet which was somewhat larger and more powerful, with demountable tips of different sizes and shapes, that could be sterilized. Sulzer constructed his magnet in horseshoe-shape with the idea of gaining something in the attraction force, but this was soon discarded. Gruening[23] constructed in 1880 a permanent magnet of a small size, useful only when in direct contact with the foreign body, such as the removal of particles from the anterior chamber. It was charged by an electromagnet or dynamo, but its force was unstable and of low degree. Then followed the magnets of Bradford,[24] Hubbell,[25] Burge,[26] Fröhlich,[27]

and many others, all based upon the same principle; that is, by varying the size of the core, the number of the windings, the diameter of the wire, and the strength of the current, a varying pulling strength was obtained.

The hand magnets most commonly in use today are those of Johnson,[28] Lippincott,[29] Hirschberg,[30] and Sweet.[31] The hand magnet of Sweet is 10¾ inches long, 2½ inches in diameter, and weighs about 10 pounds. Its core is 1½ inches thick, tapering toward one end, at which end the windings are more numerous, in a belief that a more perfect saturation is obtained. It has demountable tips of various shapes, and can be attached to the lighting circuit. Most of the modern hand magnets have a good pulling power if the foreign body is sufficiently close to the tip, *i. e.*, within 3 to 4 mm.

The first so-called "Giant Magnet" was constructed by Haab[32] in 1892 with the help of Professor Kleiners. The core of this magnet is a cylinder of soft iron, 10 cm. in diameter and 60 cm. in length, tapering at each end and arranged for detachable conical points. It is wound with a thick coil of copper wire at each end. The weight of the same is 60 kilograms. It is mounted on a wooden frame, 135 cm. high, on rollers, which is so constructed that it can easily be turned from side to side and the tip raised or lowered by tilting the magnet. The current may be supplied by the street current or dynamo. The newer model has the winding along the entire core, thereby adding to its strength.

Schloeser's[33] magnet, constructed somewhat prior to Dr. Haab's, was somewhat smaller, and wound by a single coil of copper wire consisting of some 500 turns. The core was 13 cm. long and 4 cm. in diameter. It was mounted on a wooden frame, which had attached a chin-rest for the patient.

Mayweg's[34] magnet is similar in size to Schloeser's, but has again as many windings of copper wire, and is so mounted

that by a screw attachment the magnet can be approached to the patient's eye. Other giant magnets were constructed by Schenkel[35] and Schuhman and Hirschberg,[37] built on the same lines as the foregoing.

Volkman[38] devised a magnet which differed materially in its construction. He used a long soft iron bar 5 cm. in diameter and 95 cm. long. The winding at the end used for operation was much thicker than the other end, it being 14 cm. at the former end and 10 cm. at the latter, and the core extended out from this end. By this method of construction he claimed that he could obtain the greatest magnetic force at the pole. The entire magnet was incased in leather and suspended by a metal ring. It has three demountable tips, one flat, 15 cm. in diameter, one hemispheric, and one conical. The magnet is balanced by a counterweight and is held by adjustable metal arms, which are attached to the wall of the operating room, and thus easily moved and adjusted. Volkman later modified this magnet and built them in three sizes.

Another magnet of a radically different construction was made by Mellinger.[39] This consisted of an oval (solenoid) formed by a great number of windings of thin copper wire, surrounded by a heavy broad ring of soft iron. At the lower part of the ring was a plate of ebonite, which could be raised or lowered, and was used as a rest for the operator's hand. The instrument is mounted on a heavy solid stand. When an electric current is passed through this ring magnet, a homogeneous magnetic field is established within the ring, which has its greatest intensity near the center. By holding an iron rod in this field it becomes magnetic. Rods of various shapes and sizes are used in this field, also a heavy iron beam may be attached within the ring when greater pulling power is desired. Amberg[40] and Jurnitschek[41] state that this magnet has greater strength with a lower number of ampere turns than the average electromagnet. Hallauer[42] described

a new mounting for this magnet, using the magnet suspended and the patient in the recumbent position. The interior of the ring is sufficiently large to admit the patient's head, and the advantage claimed is that the operating field is exposed to view and that the magnetic rods are more easily manipulated (some very thin) and under better control than the tips of the hand magnet or giant magnet.

Lancaster[43] recently constructed a large magnet on the Haab model, of more than average strength, based upon the number of windings, length of core, and strength of the current used. He comes to the following conclusions: "The strength of the pull depends not on the intensity of the magnetic field but on the difference in the intensity between neighboring portions of the field." Direction of pull is not necessarily along the line of force, but toward the stronger part of the field by the shortest path." "Limit of strength of pull is not the saturation point." "Cores should be ten to fifteen times as long as they are thick, and should be cylindric in shape." "Coil should be wound over the whole length of the core."

In 1914 Lancaster[44] demonstrated an instrument to test the pull of magnets, claiming this to be more accurate than the measurement by sustaining weights. He found that the pull was dependent upon the fact that a proper tip must be used, preferably a short conical one, as this style tip gave the best results. Sweet[45] tested the strength of the Haab, Hirschberg, and Sweet magnets, and found that the large magnets were decidedly superior for all splinters situated 10 mm. or farther from the magnet. When the splinter is 5 mm. distant, the drawing power of the Haab is greater, but the difference in power for this distance is not marked. At 2 mm. the Sweet magnet was superior to the Haab.

The diagnosis of foreign bodies within the eyeball has been much simplified since the perfection of the *x*-ray. However, the other methods of examination formerly used are still of

great help to us. The history of the accident or injury gives us very valuable information sometimes. These accidents occur most frequently to men, especially those employed at trades which necessitate their using tools made of iron and steel. The tools are usually made of hard steel and therefore brittle. The usual history of these cases is that, while the workman or some one near him was chipping or striking iron or some other metal with a hammer or chisel, a particle of something struck him in the eye. A history such as this, in fact, any injury to the eye of an individual working under these circumstances, should give rise to the suspicion of a foreign body. The small particles of metal which cause the trouble in these cases usually move with great velocity and sufficient force to penetrate the tunic of the globe. The cornea and sclera offer the firmest resistance, and next to these the lens. If the foreign body does not lodge in either one of these situations, it usually penetrates further, entering the vitreous, where fully 75 per cent. of foreign bodies of the globe are localized.

The character of the wound soon after injury depends upon the shape and size of the foreign body, but usually it is small and clean cut, as the particle of metal travels in the direction of its long axis. This is due to the resistance offered by the air as the body passes through space. Curved or angular wounds are very seldom caused by flying particles of metal. However, if there is any doubt regarding the possibility of the wound having been caused by a piece of metal, the *x*-ray should be resorted to at once. In those cases where the injury has occurred some weeks or months before the patient presents himself for examination the sclera may have healed over so completely that it is almost impossible to find any indication of the probable location or course of the foreign body. Sometimes, with oblique illumination, with or without the aid of a good binocular loop, a foreign body may be recognized in the cornea, anterior chamber, iris tissue, or

on the lens, when in the anterior layers. If the media are sufficiently clear, a foreign body may be seen with the ophthalmoscope in the vitreous, retina, or choroid, providing it is not too far forward near the periphery, and therefore out of the range of the ophthalmoscope. A foreign body will sometimes give a metallic luster or a glistening reflex with the ophthalmoscope if it has not been in the eye too long; for it is well known that a foreign body which has remained in the eye for any length of time usually becomes covered with an exudate which will conceal it from view, or it may be hid by an opaque lens.

Previous to the introduction of the *x*-ray for locating foreign bodies in the globe much stress was placed upon the proper ophthalmoscopic examination, by means of which it was almost possible accurately to determine the location of the foreign body with relation to the external points on the sclera.[46]

Another form of diagnosis used was that afforded by the large magnet. In this method the patient was placed before the magnet to ascertain whether or not it was possible to obtain any movement of the foreign body, as such movement usually produced pain. Pain so produced was known as "pain reaction." The great danger in such a procedure is very evident, as we had no way of telling the size or location of the foreign body, and the possibility of drawing and lodging it in the ciliary region or lens was imminent.[47] A negative result by this means did not mean much,[48] as we know that foreign bodies which have been in the eye for some time become covered with exudate and give no reaction with the magnet. Because of the foregoing facts it is easily seen why this method of diagnosis should not be attempted unless all other methods have failed.

The sideroscope is a valuable aid to diagnosis. It was Thomas R. Pooley[49] who first used the magnetic needle for detecting the presence of magnetic particles within the eye.

He demonstrated the delicacy of this method by various experiments. Then Pagenstecher and Fröhlich[50] constructed similar suspended magnetic needles.

In 1890 Leon Gerard[52] constructed his magnetometer, which he demonstrated at the Tenth International Medical Congress in Berlin. It consists of a small magnet composed of numerous steel bars, each 6 mm. long; this is suspended by a silk thread through a glass tube. The magnet is partially incased in glass. A small mirror is attached to the magnet, and this reflects a scale attached above a small telescope. The suspected eye is brought near the instrument, and if a foreign body is present, it attracts the magnet, and the change of position of the scale as reflected by the mirror is noted through the small telescope. When attraction takes place, it is called a positive reaction. This instrument did not find much favor.

The sideroscope of Asmus[53] consists of a highly magnetized needle suspended by a silk thread through a glass tube. The needle is also incased in glass, and attached to the needle is a mirror for reading the deflection upon a scale by a telescope. The eye is brought close to the needle, and the deflection gives an indication of the presence of magnetic particles within the eye. This is a delicate instrument. It is suitably mounted and its findings are very accurate.

In 1899 Hirschberg[54] constructed a simplified sideroscope, based upon the principles of the Asmus instrument. It is smaller, less complicated, and carries a needle suspended in much the same manner. Upon the mirror attached to the needle is reflected a ray of light which is directed upon a scale at some distance. The slightest deviation of the needle will cause a distinct movement of the light upon the scale.

The sideroscope can detect the presence of a magnetic particle in the eyeball, and in the hands of one with much practice its location can be suspected but never accurately

established. The sideroscope is easily influenced by electric currents. The passing of electric street-cars some distance away will cause a deflection of the needle. Koster,[55] by adjusting the position of the instrument with relation to external currents, and with the use of a small neutralizing magnet, has somewhat overcome this difficulty. In cases of foreign bodies located by the *x*-ray the sideroscope will, if there is any doubt, help solve the problem of its magnetic qualities. It occasionally happens that the *x*-ray plates fail to show a foreign body in the eye, which is later found after operation or enucleation. Sweet[56] and Randolph[57] each report such cases.

In a case at the Brooklyn Eye and Ear Hospital the *x*-ray failed to show a foreign body some 10 mm. long and 5 mm. wide and 2 mm. thick, one edge of which was curved. Repeated *x*-ray plates were taken, with negative results; the eye was subsequently enucleated for panophthalmitis, and the foreign body found in the vitreous.

Janssen[58] (Stockholm) devised an instrument called the siderophone, which consists of a magnet with an interrupter attached to a telephone. When brought in contact with or close to an eye containing a metallic body, the telephone would ring.

Weiss[59] constructed a similar instrument called the metallophone. He used a telephone, a Wheatstone bridge to measure the electric resistance, and two copper platinum-plated needles as electrodes, the latter being placed some 4 mm. apart on various points of the eye. When a metallic body was present, the telephone would ring, due to the difference in resistance between the poles.

Both instruments are of little practical value and only have historic interest.

The most accurate method for the diagnosis of foreign bodies within the eye, which cannot be detected by the customary method of examination, is the Roentgen-rays. Since

their discovery by Professor Roentgen in 1895 they have become the mainstay in diagnostic procedure.

The early development of the *x*-ray as an instrument of diagnosis for foreign bodies in the eyeball is a long and interesting chapter. Extensive references as to the progress of its development are recorded in Graefe-Saemisch Handbuch der Augenheilkunde.[60]

Such early writers as van Duyse and others claimed that the shadow cast by a foreign body within the eye would be lost in the structures of the bony orbit. However, Stern, Opitz, Dahlfeld, and others soon demonstrated that a foreign body located anywhere within the eyeball could be seen upon an *x*-ray plate. It having been proved that the foreign body could be detected on the *x*-ray plate, the next problem left to be solved was its localization. This problem was a most important one, for the knowledge of the presence of a foreign body was of little value providing some means could not be found by which it could be accurately located. This fact gave rise to several early methods of localization. One was an attempt to use the bony prominences about the orbit as guides, but it was found that the interpretation of the *x*-ray plate by means of these anatomic relations was not very successful, for the bony landmarks were usually not sufficiently shown. Then the stereoscopic method was introduced, which consisted of taking pictures at slightly different angles. They were then mounted in a stereoscope to give the proper sense of depth to the pictures. By this means the position of the foreign body could be relatively established, but not accurately located.

Later, more exact methods of localization were devised, all of which depended upon geometric measurements for their accuracy. It was found that, knowing the position of one or two fixed points, the distance of the source of light from the *x*-ray plate, and in some cases its direction or angle, the foreign body which produced the shadow could be ac-

curately placed in space, and its relation to the known points established. To Sweet and Mackenzie Davidson the greatest credit is due for devising and perfecting these methods of localization.

Sweet,[61] in 1897, described his method. He placed the patient in a recumbent position and used a localizer with two points. One was placed at a position above and near the cornea, the distance having been ascertained by measurement, and the other was placed on the same plane, being at a known distance from the first. Two pictures were then taken—one in the horizontal plane and the other at a little distance below this plane. It was found that it was important to have the axis of the eyeball and the indicator parallel to the plane of the plate. He strapped the plate-holder, with the indicators attached, to the head of the patient, so that the picture showed both of the indicators and the foreign body, and, knowing the position from which the picture was taken, the indicators could be easily differentiated. The measurements were then taken upon the plate and transferred to an especially designed chart. By this means he was able to ascertain at his final reading the backward, horizontal, and vertical displacement as measured from the center of the cornea.

In 1909 Sweet[62] presented a modification of his former method. At this time he had the tube-holder, the indicating ball, and the plate-holder placed upon a movable stage, thus retaining them in constant relation to each other. One indicator was used, as the direction of the rays was known. He took two exposures upon the same plate, using half of the plate for each exposure. This is a very valuable procedure, as the patient is less liable to move his position, since the time required for taking the *x*-ray is reduced almost two-thirds. After the plate is developed, it is placed in a frame against a key-plate, and a reading of the position of the foreign body is taken and transferred to a chart, as in his first

method. The apparatus for this method is ingenious, yet simple, and the means of localization is accurate and requires no mathematical calculation or geometric drawings.

The Mackenzie-Davidson method[63] divided the plate into four parts by means of crossed wires, an imprint of the wires being made upon the patient's skin, and the centers of these wires was definitely adjusted with the center of the anode of the tube. Two plates were then exposed at a definite distance above and below the line of centering—to this line was given the name zero. The developed plates were then placed in an especially devised machine in the same relation as they were when they were exposed. Silk threads were then stretched in the direction of the rays of light as they emanated from the anode of the *x*-ray tube to the shadow on the plate. The crossing of the threads indicated the location of the foreign body. This position was accurately measured and deductions were made with reference to the markings on the patient's skin. This method is adaptable to any part of the body.

Hullen[64] modified this method. He also used crossed wires which were centered at a definite distance from the anode. Two positions were taken—one in the horizontal plane and the other at a definite distance below it. The indicator was then placed at a known distance from the cornea. The results were graphically drawn on a plotting board by lines representing the shadows of the indicator and foreign body in the rays of light. This dispensed with cumbersome apparatus, was quicker and more accurate. The deductions could be easily obtained, and the results placed upon an especially arranged chart.

The rectangular or graphic method consisted in taking the pictures at right angles, one anteroposterior and the other from the side. Crossed wires with definite relation to the cornea and temple were used for localizing points. This method, somewhat modified, is advocated by Fox.[65] Holth,[66]

of Christiania, used this method, but in order to obtain more points for localization, attached small buttons of lead to the conjunctiva above and below the corneal margin.

Grossman[67] used for his method of localization the displacement of the shadow of the foreign body by making pictures in extreme adduction, abduction, elevation and depression of the eye, the pictures being taken from the same position.

The method of Ramsay,[68] a modification of the Mackenzie-Davidson method, and also that of Guilloz,[69] are based upon the same general principles, but require different mathematical calculations to obtain the results.

The method used at the Brooklyn Eye and Ear Hospital, and the one with which I am the most familiar, is that of Dixon.[70] Its results have been most satisfactory. This is a modification of the preceding methods, with some original features. As in the Mackenzie-Davidson method, the exact distance from the plate to the anode is known, and two pictures are taken from known positions above and below the horizontal plane. A single ball indicator is placed anterior to the cornea at a known distance, as in Sweet's method. This indicator is on an adjustable extension attached to a head-band about the patient's head, thereby avoiding any injury to the eye in the event of the patient's moving his head. The patient is placed in a recumbent position, with his head steadied by side-pieces and grasping an adjustable bar between his teeth, and the visual axis is held parallel to the axis of the plate by fixing his gaze upon a suspended ball above the eye. The instrument is built upon a table, and the *x*-ray tube is easily raised or lowered by a screw device. The position of the foreign body and the indicator are measured upon the *x*-ray plates in relation to the crossed lines, and the measurements recorded on a plotting board which has lines drawn to represent the lines of shadow, as in the Hullen method. The plotting and interpretation are simple, rapid, and most accurate.

There is with this method, as there is with all other methods, one disadvantage, and that is that only two dimensions of the foreign body can be ascertained, thereby making it impossible to estimate the exact size. This can be overcome to a great extent by taking another exposure at right angles.

The localization of the foreign body is estimated from the center of the cornea, as shown by the indicators, and deductions are made therefrom. The charts used in recording the location of the foreign body assume the eye to be about 24 mm. in the anteroposterior diameter. In eyes of exceptional length, axis myopia, or eyes elongated by disease, and in flat eyes, as high hypermetropia or phthisis bulbi, an error could easily be made as to location of a foreign body, especially in relation to the outer tunics of the eye.

No attempt should be made to extract a foreign body from the eyeball until the exact location has been ascertained. As to the method of procedure, I might say that it is impossible to follow any general rule, for each case must be considered individually and treated according to the conditions present. However, the main factor to be considered is to choose the method which will cause the least possible trauma, and avoid, as far as possible, any entanglement of the foreign body in the tissue of the iris and ciliary body. In recent cases, seen within six hours after the accident, and where the wound of entrance is still open, it is considered advisable to try and withdraw the foreign body through the wound of entrance. When the wound has closed, as is the case in most of the patients seen some time after the accident has occurred, it is necessary to make a new opening for the extraction. The location of the new incision, of course, is always dependent upon the situation of the foreign body and the method to be used for its extraction. If it is in the iris or lens, there is no question as to the advisability of drawing it forward into the anterior chamber and then making an open-

ing through the cornea for the insertion of the tip of the magnet, preferably the hand magnet, as it is more easily manipulated. With the proper current and a magnet of a standard make the foreign body can usually be extracted without much difficulty. But when the foreign body is in the vitreous, where about 75 per cent. of them are found, the procedure varies. Some operators, since the development of the large magnet, especially the Haab magnet, advocate drawing the foreign body forward through the vitreous around the lens, through the suspensory ligament to the back of the iris, and thence through the pupil into the anterior chamber. Other operators, and especially Sweet, follow the method as advocated by Hirschberg, *i. e.*, to open the eye through the sclera to the vitreous and draw the foreign body out through the incision. This is known as the posterior method.

The anterior method, as outlined by Haab,[71] is as follows: The eye is cocainized and cleansed with all the necessary aseptic precautions. The patient is then placed before the stationary Haab magnet, his head being controlled by the hand of the operator, who stands to the side, in front or in back of the patient. When a small or medium-sized foreign body is present, the center of the cornea is placed exactly opposite the pole of the magnet, and the patient is told to look directly at the tip of the magnet. If the foreign body is large, the procedure is changed, in that the eye is not brought so close to the tip of the magnet, but the center of the cornea is kept in relatively the same position. Upon turning on the current the foreign body may be seen to appear behind the iris. In the event of this not happening, the current is repeatedly turned on and off. If no bulging of the iris takes place, the eye should be turned slightly to the side so as to bring the more lateral portions of the cornea opposite the tip of the magnet, care being exercised not to draw the foreign body into the ciliary region. When the iris bulges in its periphery, the magnet must be so ap-

plied that, by changing the axis of the eye, the foreign body can be drawn through the pupil into the anterior chamber. If the foreign body is smooth, this can usually be accomplished with comparative ease, but if it is large, with a roughened surface, injury to the lens and entanglement with the iris are often difficult to avoid.

After the foreign body is drawn into the anterior chamber it is removed through a corneal incision. Haab often uses his large magnet for extracting the foreign body from the anterior chamber, but most operators prefer the small points of the hand magnet.

There have been minor modifications made in this general technic, depending on complications and the choice of the operator, but the general procedure has been little altered. When entanglement with the iris is threatened, an iridectomy is usually advisable.

The posterior method, as practised by Hirschberg,[72] was as follows: The patient was given a general anesthetic and the eye turned and held by a suture placed through the conjunctiva near the corneal margin. A large conjunctival flap was made, including some of Tenon's capsule, over the proposed site of the incision. A meridional incision was then made, some 5 to 7 mm. long, through the sclera, near the equator, below and to either side of the inferior rectus. The sclera was punctured with a double-edged Graefe knife, the opening being enlarged as required. The incision was then held open with a small tenaculum, and one of the large tips of the hand magnet placed against the incision; if the foreign body was not extracted, one of the smaller tips was used, and this entered into the incision in the supposed direction of the foreign body. I say supposed direction of the foreign body, because most of Hirschberg's cases were not localized by *x*-ray. The instruments he used, with the exception of the magnet, were made of non-magnet material. If the vitreous prolapsed, it was cut off, and the incision closed by

suturing the conjunctival flap with three silk stitches. The patient was usually kept in bed for one or two days, but remained in the hospital for one or two weeks.

With the exact methods for localization now at our command it is usually possible to open into the vitreous at almost the exact point, or very near the location of the foreign body, thereby lessening the trauma.

Jackson[73] has devised a method to be used when the foreign body cannot be removed, due to its being encysted by exudate. He introduces the point of a sharp cutting scissors into the incision, the scissors being in contact at its joint with a strong hand magnet. With the current turned on, he makes repeated short cuts into the underlying tissue in the direction of the foreign body, thus opening a path to the same. He reports several cases with successful results. In one case I used this procedure and removed a foreign body which could not be removed with the magnet alone.

The question as to which method, the anterior or the posterior, offers the best results in the removal of foreign bodies from the vitreous is undecided. There are ardent adherents of each method, their opinions being justly based upon results obtained by long and rich experience. No doubt both methods give their results and also their failures, the failures often being caused by circumstances beyond the control of the operator. Those who give preference to the posterior method claim that this method produces less trauma to the eye, and when properly carried out, is comparatively simple and usually successful, as there is no danger of injuring the lens or entangling the foreign body in the iris or ciliary body; also that by this method the foreign body need not make a second long excursion through the eyeball in being extracted.

The advocates of the anterior method also claim that by this method the trauma is less, and the danger of infection not so great as by the posterior method, when the vitreous has been opened. They further claim that corneal wounds heal

more readily than incisions of the conjunctiva and sclera, and that the danger of choroidal hemorrhage, retinal detachment, and interference with the nutrition of the vitreous is much less. Personally, I have had the best results with the posterior method, and in the five cases herewith reported this method was used.

The claim that infection occurs more frequently in cases treated by the posterior method is not borne out by the cases reported in recent years. I believe that, to avoid infection, a good-sized conjunctival flap should always be made and subsequently sutured, care being taken that the sutures are not tied over or near the scleral opening. The conjunctival flap is usually closed by exudate within several hours, and suturing of the sclera is not necessary. There was no prolapse of vitreous in any of my cases, thereby lessening the trauma, and I attribute this, to a great extent, to the fact that I used a small incision. Before making the incision, the location should be most carefully planned and not guessed at at the time of operation. The conjunctival vessels give good landmarks, and a sterile compass is a valuable aid. The incision should be made with a Graefe knife or one of similar shape, a puncture being made through the sclera, care being taken not to enter the vitreous more than 1 or 2 mm., the scleral puncture being enlarged as the knife is withdrawn. I usually make a T-shaped incision, the first or vertical incision about 3 to 5 mm., and the second or horizontal incision from 2 to 3 m., this latter incision being made as far as possible without entering the vitreous.

Plunging the knife through the sclera into the vitreous is bad practice. The lips of the incision should be held apart by small retractors, and the tip of the magnet should be introduced between them, never into the vitreous at the first attempt, and never thrust into the vitreous. If the incision is properly located, the foreign body can usually be extracted at the first attempt and without much trauma. When the

foreign body does not appear on the first attempt, I then use smaller tips and advance through the incision in the direction of the foreign body, and in none of my cases was it necessary to enter the vitreous more than 2 or 3 mm. Jackson's method was very successful in the case in which it was used by me.

When retinal detachment occurs as a result of the operation, I believe it is due to undue trauma by making the incision too deep or by entering the tip of the magnet too far into the vitreous and moving it about with undue caution. Sweet states that he has never had a retinal detachment due to operation. Where the proper care is taken and retinal detachment does occur, I believe it to be due to the injury produced in the vitreous by the foreign body at the time of its entrance. In some cases a distinct line can be seen in the vitreous, corresponding to the tract made by the foreign body. This was present in one of my cases, and I believe it was undoubtedly due to the injury, for a small band of connective tissue could be seen extending from the original wound to a point where the foreign body had ruptured the retina and choroid on the opposite side of the eyeball.

In one of my cases with a scleral wound I was able to extract the foreign body through the wound of entrance, and in one other, in which I tried to extract through the original wound located at the limbus, complete traumatic aniridia resulted. Dr. Hiram Woods,[74] of Baltimore, reports one similar case.

I should consider it advisable to work with a large magnet suspended by a counterweight, and not one in a stationary position, such as I have used. A foot control for the current, so that it may be regulated by the operator, seems to me would be much better than having the current controlled by an assistant.

The following ten cases came under my personal treatment during the past five years:

T. C., boy aged thirteen years, brought to my office November 18, 1911. On November 11th, while striking a nail with a hammer, something flew into his right eye. A sharp pain followed, and he was taken to the family physician, who ordered an eye lotion. Two days previous to my examination vision became blurred and the eye red and irritable. Left eye normal, V. = 6/6. The right eye showed marked conjunctival and ciliary injection, with a small 2×2 mm. granulated area in the conjunctiva covered with some fibrinous exudate, 8 mm. to the nasal side from the limbus and 2 mm. below the horizontal plane. The pupil was round, reacted well, and the lens was clear. Vitreous was full of hemorrhagic streaks which seemed to radiate to the side and site of injury, and there were also some fine, dust-like opacities. V. = 6/60. The pupil was dilated with atropin, but no foreign body could be seen. An *x*-ray picture taken November 21st (delay due to patient) showed a piece of steel, the anterior end 11 mm., the posterior end 13 mm., back of center of cornea; 6 mm. below the horizontal plane; 1 mm. on the temporal side of the vertical plane.

Operation November 22d, under ether anesthesia. Triangular conjunctival flap was made over the location of the foreign body, and a 3×2 mm. crossed incision was made through the sclera. The short conical tip of the Hirschberg hand magnet was inserted into the wound, but not more than 2 mm. After a few attempts, the wound being held open by little hook retractors, the foreign body was easily withdrawn. It measured 3×1.5×0.5 mm. It was sharp, but had no rough edges. The conjunctival flap was sutured with two silk sutures. The wound healed promptly, and on the fourth day the stitches were removed. The patient remained in the hospital five days. November 28th: Lens clear, vitreous less cloudy, hemorrhagic streaks less. V. = 6/24.

December 8th: Vitreous clearing, fundus details visible, thick streak of exudate extending into vitreous from site of original injury. A linear tear in the choroid and retina, about one-half disc diameter in length. Fine deposits on posterior lens capsule, especially below. V. = 6/12.

December 23d: Pupils now equal size; exudate in vitreous has shrunken. V. = 6/9+.

May 16, 1914: V. O. D. with sph. +1 D. = 6/6. No vit-

reous dust. The haziness on the posterior lens capsule is no longer to be seen. The exudate in the vitreous is limited to the original site of injury extending to tear in retina.

February 27, 1916: V. O. D. = 6/6+. Field of vision normal. No external scar visible at original site of injury, only where sclera had been entered at the time of operation. Extending backward a thin band of connective tissue connected the wound of entrance with the tear in the choroid and retina. Pigment along the margin of the tear. The incision through the retina and choroid made at the time of the operation is clearly visible in the extreme periphery. No pigment at these margins and no exudation into the vitreous from this point.

Dr. W. W., aged thirty years. On May 13, 1913, at 10.30 A. M., while using a hammer to strike a hatchet in wedging a board, a chip of steel struck the right eye. He stated that he had no pain, only the sense of impact, with a flash of bright light and the appearance of numerous dark spots before the eye. He arrived at my office at 11.40 A. M.

Left eye, V. = 6/6. Right eye showed a sharp, clean cut through the conjunctiva and sclera on the nasal side, running obliquely upward, 4 mm. in length, with its inner edge 1 mm. from the limbus in the horizontal plane. Diffuse hyphemia filling one-third of the anterior chamber. Pupil dilated slowly under atropin. Hemorrhage in the vitreous toward the nasal side. *X*-ray showed a foreign body suspended in the vitreous.

Under cocain anesthesia the patient was placed before the Haab magnet, in such a manner as to exert a direct pull in line of the wound of entrance and progress of the foreign body. The body was easily extracted, coming out promptly through the wound of entrance as the eye was approached to the magnet. The conjunctiva was then drawn over the wound in the sclera and sutured. The operation was done three and a half hours after the accident.

The patient remained in the hospital one week, and the conjunctival sutures were removed on the fourth day.

May 24th: V. O. D. = 6/12, with sph. + 75 ⌒ cyl. + 0.50. 15° = 6/9. Eye showed no reaction except at site of injury. The patient was seen at intervals until February 2, 1914, and at this time vision with correction was 6/6.

February 25, 1916: The right eye, except for a small healed scar to the inner side of the limbus, appears normal. No vitreous opacities. Fundus normal. V., with cyl.+ 0.50 ax. 15° = 6 4.5.

G. J., aged fifty-six years, ironworker, was injured in right eye December 24, 1913, while striking a steel die with a hammer. Some forty-eight hours later he came under my observation. The left eye normal, V. = 6/60, compound hyperopic astigmatism. Right eye showed, 3 mm. above and 3 mm. to the nasal side of the center of the cornea, a small perforating wound which had healed. Above and slightly to the nasal side of this was a small hole in the iris. On dilating the pupil lens opacities in the upper nasal quadrant were seen, also fine, feather-like opacities under the lens capsule. No entrance wound of foreign body into lens could be seen. X-ray picture showed a foreign body 12 mm. back of the center of cornea, 3 mm. below the horizontal plane, and 10 mm. to the nasal side of the vertical plane.

Under cocain anesthesia a triangular conjunctival flap was made. An incision some 3 mm. in length, with a shorter one at right angles, was made in the sclera near the location of the foreign body. The tip of the hand magnet was introduced between the lips of the scleral incision. On the second attempt the foreign body appeared in the wound, drawing the choroid up before it. The choroid was incised with scissors and the foreign body removed. The piece was 3 × 1.5 by 0.5 mm. It was a thin scale of steel with no rough edges. Sutures removed the third day.

Left hospital January 2d. The lens cloudiness had not increased. There were some fine vitreous opacities. V. = 6 24. This eye was always the weaker, due to 5 D. of astigmatism uncorrected. Patient was seen from time to time. The cloudiness under the lens capsule disappeared, but there were still some opaque streaks in the periphery of the lens. The vitreous opacities had disappeared.

July 12, 1915, one and one-half years after accident and operation, the pupil of right eye was somewhat larger than left, the lens opacity localized to upper nasal quadrant had not increased, and vision, with correction, equaled R. E., 6/12; L. E., 6/9+.

February 27, 1916, pupil R. E. 4.6 mm.; L. E., 3.5 mm.,

reacts promptly. Small horizontal scar in cornea visible. Small hole in iris still present, but no exudation over it. In the extreme periphery of the lens, upward and inward, the finest net-like opacity was seen. No local intra-ocular lesion found; no vitreous opacities. V., with correction = 6/12. Since this was always somewhat of an amblyopic eye, the vision might be regarded as normal for such an eye.

B. G., engineer, aged thirty-three years, was injured in left eye October 13, 1914, while striking a vise with a hammer. Vision immediately became blurred. He came under my observation twenty-four hours later. V. R. E. = 6/6. Left eye, slight ciliary injection, abrasion with cut in the cornea (now closed) 2 mm. long in the upper nasal quadrant, and about 2 mm. from the limbus. A slight tear in the iris immediately under the corneal wound. Pupil small and round and reacted to light. Tension normal. Lens opacity in the upper nasal quadrant, and stellate opacity in the posterior layers. Atropin dilated pupil evenly; no foreign body could be seen in fundus. Above and to the nasal side, bright red hemorrhagic reflex; some thick vitreous opacities. *X*-rays showed a foreign body in the vitreous, 11.5 mm. back of the center of cornea, 0.5 mm. to temporal side of vertical plane, and 10 mm. below the horizontal plane.

The following day, under cocain anesthesia, an attempt was made with the Haab magnet to draw the foreign body forward. Magnet was used in the customary way, and repeated attempts to extract the foreign body failed. No pain reaction was felt. The posterior method was then tried. Under a carefully dissected conjunctival flap a crossed incision of sclera was made; and the tip of a hand magnet (Hirschberg) was then introduced between the lips of the incision. The foreign body was easily removed after several attempts, care being exercised not to enter the wound more than 2 or 3 mm.; flap was then sutured.

On the following day a severe iritis developed, and the vitreous became cloudy. There was a specific nose condition, perforated septum, and saddle-nose. The Wassermann was positive. Under rigid antisyphilitic treatment the inflammation slowly subsided, and the patient left the hospital in a week. Two weeks later all external reaction had subsided. There were a few pigment deposits on the lens

capsule. The lens cloudiness was increasing; the vitreous opacities were somewhat less dense. Tension normal. V. = 1/36.

Two months later eye appeared normal externally. Pupils are of equal size and react promptly. V. = 1/12. Vitreous still filled with many dust-like and larger opacities. The field of vision was not constricted. The lens opacity was increasing; the eye had assumed a position of divergence. The patient was still taking a mixed treatment. Almost a year elapsed before the patient was again seen. The eye at this time was white; pupils of equal size. Lens opaque in its posterior layer. There was a still marked iris shadow. Patient complained of floating spots before this eye. V. = hand movements; light projection good in all directions. Tension normal. The cataract might be sufficient to reduce the vision, but no doubt choroiditis and vitreous changes are present, based upon the specific history.

E. S., aged forty-eight years, ironworker, came to the Brooklyn Eye and Ear Hospital September 26, 1914, and stated that three days previously, while striking a block of iron with a hammer, a chip of steel or iron flew into his left eye. He gave a history of experiencing a sharp pain at this time, and his vision became cloudy. V. = 6/60.

The eye showed a moderate reaction, and on the cornea could be seen a small healed wound, 1 mm. in length, 3 mm. from the limbus, a little to the nasal side of the vertical meridian. Slightly higher and to the nasal side a small tear in the iris could be detected. The pupil reacted promptly, but the lens was cloudy in the upper half, especially in the deeper layers. The vitreous was full of opacities, but no foreign body could be seen. The *x*-ray plate showed a foreign body 3.5 mm. long. Anterior end 10 mm., posterior end 13 mm., back of the center of the cornea; anterior end 9 mm., posterior end 10.5 mm., below the horizontal plane; anterior end 8 mm., posterior end 8.5 mm. to the temporal side of the vertical plane.

The patient's eye was cocainized and he was placed in front of the large Haab magnet, but after repeated and persistent attempts we were unable to extract the foreign body.

Another *x*-ray was taken, but the restlessness of the patient caused the plotting to be incorrect. In view of the

previous *x*-ray, the patient was prepared for operation, and after dissection of a conjunctival flap a crossed incision, 3×1.5 mm., was made near the location of the foreign body as outlined in the first plotting. The tip of a Hirschberg hand magnet was then introduced through the incision, and repeated attempts made to extract the foreign body, but without success. A strabismus hook, magnetized by contact with the magnet, was then introduced in the direction of the foreign body, but with no results. At no time during the operation was there any "pain reaction." The conjunctival flap was sutured and the eye kept under observation.

On October 16th the patient left the hospital. At this time the lens cloudiness was increasing, and vitreous opacities were numerous. V. = finger at one meter.

The patient was not seen again until December 7th, when he returned to the clinic. At this time another *x*-ray picture was taken and the center of the foreign body localized—7 mm. back of the corneal center, 2 mm. to temporal side of the vertical plane; 12 mm. below the horizontal plane. During the exposure the patient was extremely irritable and restless.

Upon examination of the eye the lens appeared almost completely opaque; pupil reacted promptly; no signs of siderosis. V. = hand movements.

Patient was advised to have another *x*-ray picture taken and to remain in the hospital for observation, but he did not return.

The failure to obtain the foreign body in this instance by the anterior method was probably due to the insufficient strength of the magnet. The failure of the posterior method was probably due to faulty localization, and I may have failed to enter the sclera at the proper place. Again, the foreign body may have been of a non-magnetic material, or it may have become encysted by exudate.

G. K., chipper helper at the ironworks, while striking with a chisel and hammer on July 22, 1915, a chip of steel flew into left eye. He was attended by a local physician. On July 24, 1915, he was seen at my office. Right eye normal; V. = 6/6. Left eye, moderate reaction. A 3 mm. ragged wound in the sclera, and extending 1 mm. into the cornea on the nasal side

in the horizontal meridian, was visible. Pupil drawn slightly to this side, iris incarcerated, and at base is torn and ragged. Lens cloudy on the nasal side. Small hyphemia. *X*-ray plates showed a foreign body 2.5 by 1 mm., located 11 mm. back of the center of cornea, 7.5 mm. below the horizontal plane, and 1 mm. on the temporal side of vertical plane.

Patient would not consent to enter the hospital until July 27th. Under cocain anesthesia an attempt was made to draw the foreign body through the still gaping wound of entrance, with a large stationary Haab magnet, the patient's head being supported by the operator's hand, and so turned as to bring wound, magnet tip, and foreign body into a straight line. The head was several times brought slowly near the magnet without any reaction or pain. About the fourth attempt the patient brought his head suddenly forward, and experienced sudden pain as the magnet touched or nearly touched the wound. As soon as he felt pain he pulled his head suddenly backward. The current was immediately turned off, and the foreign body was found adhering to the tip of the magnet.

The body, on being drawn out, became entangled in the iris as the head was pulled backward, causing almost a complete aniridia. An extensive hyphemia developed, and some bleeding from the wound followed. The eye was bandaged, and the patient put at rest in bed.

The foreign body was much larger than the *x*-ray showed it to be, and had many rough edges and sharp points. It measured $5 \times 2.5 \times 1.5$ mm.

A rather intense reaction followed, lasting several days. The hyphemia was slowly absorbed and the wound closed. A small strip of iris remained near the wound below. There was some iris pigment on the lens-capsule on the inner side. Many vitreous opacities, so no part of the fundus could be seen. V. = 1/36. On leaving the hospital in three weeks the eye was white, but the lens was becoming more opaque.

September 8: Lens cloudiness is spreading; many vitreous opacities. V. = fingers at one meter.

O. F., aged thirty-six years, steam engineer. On August 27, 1915, while working on some machinery, something struck his left eye. There were sharp pains and burning sensation in the eye. On August 30th he consulted me at my

office. Some conjunctival and ciliary injection, a small healed scar 1 mm. in lower outer quadrant of cornea and 2 mm. from the limbus, and a hole 1 mm. in diameter in the iris, somewhat above the horizontal and near the median line of the eye. The pupil reacted promptly, but on dilatation a foreign body was seen in the lens, a little to the outer side of the median line in the horizontal meridian, extending backward to the nasal side some 3 or 4 mm. A bright glistening reflex was obtained, especially from the anterior portion of the foreign body. The *x*-ray showed a foreign body in the lens; length, 4.5 mm.

On August 31st the patient entered the hospital, and under cocain anesthesia the foreign body was drawn with a Haab magnet into the anterior chamber, falling to the bottom. He was then placed on the operating table, and a paracentesis was done at the lower margin of the cornea. The foreign body, caught in the iris tissue, was disentangled by means of a spatula, and the tip of the Hirschberg magnet was introduced into the anterior chamber and the foreign body promptly withdrawn. It measured $4 \times 1 \times \frac{1}{2}$ mm.

Two days later a severe iritis developed, which was controlled by atropin, dionin, hot applications, salicylates, diaphoresis, catharsis. The patient also had a few bad teeth and some pus-pockets in the upper gum on this side, which probably influenced the iritis. In three or four weeks the inflammation subsided. A firm adhesion has formed where the iris had originally been perforated with the ruptured lens-capsule. The lens had become cloudy, but no traumatic swelling resulted. The wound in the capsule remained closed.

On October 15th the opaque lens was extracted with iridectomy above, and the patient had an uneventful recovery. Shortly after the operation a marked convergent squint developed in the left eye. This promptly disappeared, and also the diplopia after several days, upon wearing his correction: R. E. = sph. $+2 \bigcirc$ cyl. $+0.50$ ax. $90° = 6/6$; L. E. = sph. $+12 \bigcirc$ cyl. $+1.50$ ax. $80° = 6/6$.

G. W., aged thirty-eight years, ironworker. On December 30, 1915, was struck in right eye by a chip of steel from a steel bar which he was hammering. Consulted an oculist, who removed several small particles from his cornea and

ordered atropin. The sight in the eye became cloudy, and he came to my office January 16, 1916. V. L. E. = 6/21, amblyopic. Right eye, moderate reaction; 1 mm. from the nasal side of limbus a small 2 mm. vertical scar had healed under conjunctiva. Pupil dilated (atropin); lens clear; many thick vitreous opacities. To the temporal side, far forward, a large hemorrhagic area. Outline of nerve vaguely seen. Intra-ocular foreign body suspected. *X*-ray picture showed foreign body 8 mm. back of center of cornea, 8.5 mm. below horizontal plane, and 6 mm. to the nasal side of vertical plane.

On January 19th, under cocain anesthesia, a 4×6 mm. flap was dissected upward, its base toward the cornea. With a Graefe knife a puncture of the sclera was made, forming a crossed incision 3×2 mm., 5 mm. from the limbus. This incision was made on a line corresponding to five o'clock on a dial. The tip of the Hirschberg hand magnet was then introduced, and repeated attempts to remove the particle with a flat, elongated tip failed. A short conical tip was then used, and the foreign body promptly came into the wound and was removed. The tip was not introduced into the wound more than 2 or 3 mm. The foreign body was 3.5 ×1 mm. The patient remained in the hospital one week. The conjunctival flap healed promptly, and the suture was removed on the fourth day. There was almost no reaction. No fresh hemorrhages could be seen as a result of the operation. The vitreous became clearer.

January 29th only local reaction where conjunctival flap was made. Vitreous was clearer, but there were many opacities; the nerve was clearly seen. Two disc diameters to the nasal side and one disc diameter below was a local hemorrhagic area with sclera showing through the edge, probably a cut made by the foreign body before it bounded back into the vitreous. Under the lens-capsule to the nasal side in the periphery were net-like opacities (seen by loop). V. = 6/12.

February 23d: Eye white; pupil reacted promptly; the lens haziness was no longer seen. There were many vitreous opacities, and the hemorrhagic area was still present. The hemorrhage about the tear in the retina and choroid was absorbing. More sclera visible; no pigment was pres-

ent. V. O. D. = 6/6. The field of vision taken this day was normal.

J. G., aged twenty-six years; first came under my observation May 24, 1915. Stated that in August, 1912, while attempting to straighten a piece of tin by striking it between two hammers, something flew into right eye. He went to a clinic at a general hospital and was given treatment there. He saw several dark objects floating before his eye. After two months he was sent to the B. E. E. Hospital, where an *x*-ray picture gave negative results. He continued his treatment, which consisted of atropin and hot applications. The eye reaction subsided, but the floating material before the eye continued.

Twenty-one months after the accident the right eye showed no signs of inflammation. The lens was somewhat cloudy near its posterior pole and in the periphery. The iris was the same color as the other eye and reacted promptly. On or under the lens-capsule a few light brown circular spots suggested a beginning siderosis bulbi. There were no Descemet's deposits; the fundus appeared normal; no detachment and no vitreous opacities. V. = 20/70, the reduced vision being due to the lens haziness. There was no scar of the injury visible. The left eye was normal.

Although there was a history of a negative *x*-ray, taken by a competent radiographer some twenty-one months previous, as a matter of routine the patient was placed before a large Haab magnet and several unsuccessful attempts were made to extract a foreign body.

Patient returned on January 29, 1916, eight months later. He stated that he could only see light with the right eye. V. = hand movements. The lens was now completely opaque. On the lens capsule, midway between the center and periphery, numerous circular, light brown deposits were arranged in the shape of a ring in or under the lens-capsule. The iris was discolored, dark brown, with a reddish tint. The other iris was light brown. A case of siderosis bulbi, probably due to a retained foreign body. A radiograph taken showed a foreign body 9 mm. back of the center of the cornea, 10 mm. below the horizontal plane, and 3 mm. to the nasal side of the vertical plane. The patient was again placed before the Haab magnet, but with no result.

On February 11th, under cocain anesthesia, a conjunctival flap was made 4 mm. below the corneal margin and 3 mm. to the nasal side; a crossed incision was made in the meridional line. Thin watery fluid exuded from the eyeball—some five to ten drops. The tip of the hand magnet was introduced between the lips of the wound repeatedly, but with no results. The incision was enlarged some 2 mm., and various tips were introduced a little deeper, but with no results. The dull-pointed scissors were attached to the magnet (as advised by Jackson); careful and repeated small cuts were made into the tissue under the incision; the vitreous was entered some 3 or 4 mm. After repeated attempts the scissors were withdrawn, but no foreign body appeared. The magnet tip was again inserted, and upon being withdrawn, the choroid came up into the wound, with the foreign body retained under it. It was difficult to disentangle the foreign body from the choroid, as no undue traumatism was desired. The foreign body was 1.5 mm. $\times$1 mm. at its widest points, very ragged, and the edges crumbled away in the hand, leaving it about half its original size. There was practically no reaction. Tension remained below normal for several days. V. = hand movements; light projection good.

On February 21st a combined lens extraction was done, the eye healing with little reaction.

March 11th: No signs of inflammation. V. = sph. +11.0 $\bigcirc$ cyl. +1 axis 180° = 6/9. Field normal; some vitreous opacities.

E. S., aged thirty-eight years, iron riveter. On February 11, 1916, was riveting a tank-top with a hydraulic riveter when the rivet slipped, and something struck right eye. The accident occurred at the Navy Yard, and he went to the Naval Hospital. The eye was bleeding; he had severe pain and a dark haze before the eye. Went to the B. E. E. Hospital February 12th, and an *x*-ray showed a foreign body, anterior end 10 mm., posterior end 11.5 mm., back of the center of the cornea; anterior end 11 mm., posterior end 9 mm., below horizontal plane; anterior end in vertical plane; posterior end 2.5 mm. on temporal side of vertical plane. Patient transferred to my care February 15th. A perforating wound 3 mm. long was healed in the upper lid, 2 mm. above the lid margin, near the center, running diagonally

upward and inward, and a similar wound of the sclera 3 mm. long, almost horizontal, 2 mm. above the corneal margin, near the median line. Moderate reaction. Pupil semi-dilated, reacted to light; lens clear. The extensive hemorrhage and floating opacities made the fundus details indistinct.

Cocain anesthesia was used, and the patient placed before the stationary Haab magnet. As the patient's head was approached to the magnet the foreign body bulged the iris below. The foreign body was carefully drawn into the outer anterior chamber and fell to the bottom. There was some slight bleeding into the chamber. The patient walked about ten steps to the operating table and was placed in the recumbent position. On examining the eye at this time there was no foreign body in the anterior chamber, and the iris was visibly contracted to the size of a pinhead, causing the patient severe pain. This intense spasm of the sphincter irides could not be controlled by strong doses of atropin every one-half hour. The patient was placed in bed. Four hours later the pupil dilated to some 3 mm. in diameter, and the patient was again brought before the magnet, and the foreign body drawn from the posterior chamber, into which it had slipped, through the pupil without difficulty, and again into the anterior chamber. With the head bowed the patient was brought to the operating table. Through a corneal incision the tip of the hand magnet was introduced and the piece promptly removed. It measured $3.5 \times 2 \times 1.5$ mm. thick in the center, gradually tapering down toward the edges. It had sharp edges, and a cross-section would be triangular in shape. The eye showed marked reaction and was painful.

At the next dressing the pupil was not dilated more than 3 mm.; the lens showed opacities below. Some iritis present. Since then the lens has swollen rapidly and become opaque. There is no question that the sharp end of the foreign body caused laceration of the lens-capsule in the process of extraction. The man will need a cataract extraction, and I believe he will then have useful vision.

A Summary of Ten Cases of Foreign Body in the Eyeball.

Foreign Bodies in the Anterior Portion of the Eye:

One case of foreign body in the lens removed through corneal incision. Result: Cataract, which was extracted, and the patient had a vision of 6/6, with correction.

Foreign Bodies in the Vitreous:

In one case the foreign body was not obtained by either the anterior or posterior method (cause, faulty localization).

In the eight cases of foreign body removed from the vitreous the results were as follows:

One through original wound in sclera V. = 6/6, two years six months after operation.

One through original wound at limbus (traumatic aniridia) V. = fingers at one meter.

One by anterior method—large magnet (traumatic cataract) V. = fingers at two meters. Still under observation.

Five cases by posterior method:

One case, V. = 6/6, four years three months after operation.

One case, V. = 6/12 (amblyopic eye), two years three months after operation.

One case, V. = 6/6, two months after operation.

One case, V. = moving objects. Choroidal disease and cataract (syphilis).

One case, V. = 6/9, siderosis bulbi; cataract, which was extracted. Operation performed three and one-half years after injury. (Still under observation.)

REFERENCES.

1. Coulomb, C. A.: Mem. Acad. Roy. Paris, 1785, p. 578.
2. Oersted, H. C.: "Electromagnetism," Ann. of Phila., November, 1821.
3. Ampere, A. M.: Acad. Science, Paris, September, 1820.
4. Arago: Ann. de Chim. et de Phys., 1820, No. 15, p. 93.
5. Sturgeon: Trans. Soc. Arts, 1825, xlviii, p. 38.

6. Stillman's Journal, 1831, xix, p. 400.
7. Stillman's Journal, 1831, x, p. 201.
8. Weber, W. E.: Die Wellenlehre auf Experimente gegrundet, 1833.
9. Maxwell: Electricity and Magnetism, 1893.
10. Ewing, J. A.: Proc. Royal Soc., 1890.
11. Thomson, J. J.: Phil. Mag., 1897, xliv, p. 293.
12. Winkelman, A.: Handbuch der Physik, v, pt. i, Leipzig, 1905.
13. Hirschberg: "Geschichte Augenheilkunde im Altertum," Handb. v. Graefe-Saemisch, 2 ed., Leipzig, 1899, xii, p. 47.
14. Feldhaus: "Die Geschichte der Magnetoperation im Auge," Zentralbl. f. prakt. Augenheilkunde, 1903, p. 138.
15. Fabrici: Hildarci Opera, Frankfurt, 1656.
16. Morgagni: de Sedibus et causis morgarium, Helbrodenie, 1779, pt. i, p. 215.
17. Milhes, S.: 1745, "Snell," Brit. Med. Jour., July 17, 1880.
18. Himley: Die Krankheiten und Misbildungen der Auges, Berlin, 1843.
18a. "Extraction eines Stahlsplitters aus dem Auge Mittelst des Magneten," Med. Zeitung, herg. v. d. Ver. f. Heilkunde in Preussen, 1842.
19. Dixon: Ophth. Hosp. Rep., 1858, i, p. 282.
20. McKeown: "Extraction of a Piece of Steel from the Vitreous Humour by the Magnet; Recovery of Almost Perfect Vision," Brit. Med. Jour., 1874, June 20; Dublin Jour. Med. Sci., 1876, lxii, pp. 201–210; Lancet, 1878, ii, p. 253.
21. Hirschberg, J.: Berlin. klin. Wochenschr., 1879, p. 681; Zentralblatt f. Augenhk., 1879, p. 376.
22. Sulzer: Proc. of 11th Intern. Cong., March, 1894.
23. Gruening: "On the Removal of Particles of Steel from the Vitreous Chamber by Means of the Magnet," Med. Rec., 1880, xvii, p. 478.
24. Bradford: "The Electromagnet in Ophthalmology, with a Description of a New Magnet," Boston Med. and Surg. Jour., 1881, civ, p. 292.
25. Hubbell: "The Electromagnet in the Removal of Steel from the Interior of the Eye," Buffalo Med. and Surg. Jour., 1888, xxvii, p. 545.
26. Burge: "Entfernung eines Stahlsplitters aus dem Glasskoerperaum durch einem Electromagneten," Berlin. klin. Wochenschr., 1880, xliv.
27. Fröhlich: "Ein Electromagnet," Klin. Monatsschr. f. Augenheilk., January 1881.
28. Johnson: "Description of a Portable Electromagnet," Archives of Ophth., 1899, xxviii, p. 326.
29. Lippincott: "On the Advantages of Strong Portable or Easily Movable Magnets in Eye Surgery," Trans. Am. Ophth. Soc., 1900, ix, p. 152.
30. Hirschberg: Die Magnetoperation in der Augenheilkunde, Leipzig, 1899; "Mein neuer Handmagnet," Zentralblatt f. prakt. Augenheilkunde, 1901, xxv; "Über Magnetoperation in der Augenheilkunde," Berlin. klin. Wochenschr., 1907, No. 8.
31. Sweet: "Foreign Bodies in the Eye," Jour. Amer. Med. Assoc., 1902, xxxix, p. 467.
32. Haab: "Die Verwendung sehr starker Magnete zur Entfernung von Eisensplittern aus dem Auge," Berichte d. XXII. Vers. der Ophth. Gesellschaft zu Heidelberg, 1892, p. 163; 1895, p. 187; "Ein neuer Elektromagnet zur Entfernung von Eisensplittern aus dem Auge," Deutschmann's Beiträge, 1904, xiii, p. 68.
33. Schloeser: "Bedingung zur Entfernung von Eisensplittern durch den Magneten," Bericht. XXIII. Ophth. Gesell. zu Heidelberg, 1893, p. 153; "Demonstration eines verbesserten Magneten," Bericht. XXXI. Ophth. Gesell. zu Heidelberg, 1903, p. 300.
Edelman: "Untersuchungen über die beste Form des Prof. Schloeserschen Augenelektromagneten," Klin. Monatsbl. f. Augenheilkunde, 1903, xli, No. i, p. 433.

34. Mayweg: "Über Magnetoperation," Klin. Monatsbl. f. Augenheilkunde, 1902, xl, part ii, p. 1.
35. Schenkel: "Über einen neuen Elektromagneten," Deutsche med. Wochenschr., 1902, p. 924.
36. Asmus: "Mitteilungen über Schuhmannschen Augen-Elektromagneten," Klin. Monatsbl. f. Augenheilkunde, 1904, xlii, p. 241.
37. Hirschberg: "Ein neuer Eisenmagnet," Zentralbl. f. Augenheilkunde, 1904, xxviii, p. 176.
38. Volkmann: "Neuer Augenmagnet," Klin. Monatsbl. f. Augenheilkunde, 1902, xl, No. 1, pp. 113 and 353; "Ein neuer Augenmagnet," *ibid.*, 1903, xli, p. 217.
39. Mellinger: "Der Innenpolmagnet. Eine neue Verwärtung des Elektromagnetismus zur Entfernung von Eisensplitter aus dem Auge," X. Intern. Ophth. Cong., Luzern, 1904.
40. Amberg: "Weitere kasuistischer Beitrag zur Entfernung von Eisensplitter aus dem Auge mit dem Innenpolmagneten," Zeitschr. f. Augenheilkunde, 1907, xviii, p. 511.
41. Jurnitschek: "Der Innenpolmagnet," Zeitschr. f. Augenheilkunde, 1905, xiv, p. 426; also 552.
42. Hallauer: Zentralschr. f. Augenheilkunde, xxiii, August, 1909, p. 204.
43. Lancaster: "Stronger Eye Magnets," Trans. Amer. Ophth. Soc., 1915, xiv, part i, p. 168.
44. Naus: Section Ophthal., A. M. A., 1914, p. 469.
45. Sweet: "Foreign Bodies in the Eye," Jour. Amer. Med. Assoc., 1902, xxxix, p. 467.
46. Czermak: Die augenaerztlichen Operationen, 2d ed., 1908, ii, p. 652.
47. Linde: Zentralbl. f. prakt. Augenheilkunde, September, 1898.
48. Callan: "The Haab Electromagnet as a Sideroscope," Trans. Amer. Ophth. Soc., 1898, p. 456.
49. Pooley: Archiv. Ophthal., 1880, ix, p. 250; N. Y. Med. Jour., 1902.
50. Frölich: "Über den Polwechsel beim Gebrauch des Elektromagneten und über die Magnetnadel als diagnostisches Hilfsmittel," Klin. Monatsbl. f. Augenheilk., 1882, p. 107.
51. Fischer: "Extraktion von Eisensplitter aus dem Glaskörper," Arch. f. Augenheilk., xxii, p. 18.
52. Gallemaerts: "Sur la recherche des Corps etranges Magnetiques ayant perfore le Globe Oculaire avec demonstration du Magnetometer de Gerard," Soc. Franc. d'Opht., Seance du 8 Mai, p. 143.
53. Asmus: "Der Sideroskop. Ein Apparat zum Nachweis des Eisen und Stahlssplitter im Innern des Auges," v. Graefe's Arch. f. Ophth., xl, p. 280; Arch. f. Ophth., 1894, i, p. 280; Arch. f. Augenheilkunde, xxix, 1894, p. 126; "Über die genaue Lokalisation grosser eiserner stählerner Fremdkörper im Auge mit dem Sideroskop," Arch. f. Augenheilkunde, xxxi, p. 49.
54. Hirschberg: "Vereinfachtes Sideroskop," Centralbl. f. Augenheilkunde, 1899, xxii, p. 245.
55. Koster: "Ein Mittel zur Beseitseigung des stoerenden Einfluss der elektrischen Strassenbahn auf die Sideroskopie," Arch. f. Augenheilk., 1907, lix, p. 49.
56. Sweet: "Third Series of Cases of Injuries from Foreign Bodies Examined by Roentgen Rays with Results of Operations," Trans. Amer. Ophth. Soc., 1909, xii, part 1, page 117.
57. Randolph: "Two Cases in which *x*-Ray Failed to Locate a Foreign Body which was afterward Shown to be Present in the Eye," Ophth. Rec., March, 1911, xx, No. 3, p. 113.
58. Janssen: "Beal" Corps etranges magnetiques oculaires et leur extraction, Paris, 1908, p. 23.

59. Weiss: "Das Metallophon; ein Apparat zum Nachweis metallischer, auch nicht eiserner Fremdkörper im Augeninnern," Zentralb. f. prakt. Augenheilkunde, 1907, p. 100.
60. Graefe-Saemisch Handbuch der Augenheilkunde, 2d ed., iv, Part 1, p. 637.
61. Sweet: Trans. Amer. Ophth. Soc., 1897, viii, p. 91.
62. Sweet: "Improved Apparatus for Localizing Foreign Bodies in the Eyeball by Roentgen Rays," Trans. Amer. Ophth. Soc., 1909, xii, p. 322.
63. Mackenzie-Davidson: "An Apparatus for Exact Measurement and Localization by Means of the Roentgen Rays," Brit. Med. Jour., January 1, 1898.
64. Hullen: "A Simple, Accurate and Rapid Method for Localizing Foreign Bodies in the Eye," Jour. Amer. Med. Assoc., 1904, xlii, p. 881.
65. Fox: Practical Treatise on Ophthalmology, New York, 1910, p. 508.
66. Holth: "Bidrag. til Roentgenlokalisation af Okulaere Fremmedlegemer," Norsk. Mag. f. laegev., viii, 1905.
67. Grossman: "Localization of Foreign Bodies in the Eye by *x*-Rays," Liverpool Med. and Surg. Jour., 1899, xix, p. 359.
68. Ramsay: Eye Injuries and their Treatment, Glasgow, 1907, p. 84.
69. Encyclopedie Français d'Ophtalmologie, Paris, 1905, iv, p. 773.
70. Dixon: "On the Localization of Foreign Bodies in the Eye and Orbit," N. Y. Eye and Ear Infirmary Rep., 1906, xii, p. 12.
71. Haab: "Über die Anwendung des grossen Magneten bei der Ausziehung von Eisensplittern aus dem Auge," Zeitschr. f. Augenheilkunde, 1902, vii, No. 6, p. 587; "The Removal of Foreign Bodies from the Eye," Jour. Amer. Med. Assoc., 1902, xxxix, p. 463; Arch. f. Augenheilk., 1913, lxxvii, p. 271.
72. Hirschberg: Die Magnetoperation in der Augenheilk., Leipzig, 1899, 2d ed.; "Über Magnetoperationen in der Augenheilk.," Berlin. klin. Wochenschr., 1907, viii.
73. Jackson: "Scissor-Magnet Extraction of Iron from Eyeball," Jour. Amer. Med. Assoc., 1909, lii, p. 1985.
74. Woods: "A Case of Foreign Body in the Fundus; Extraction by Magnet. Complete Post-Operative Aniridia," Trans. Amer. Ophth. Soc., 1907, xi, part ii, p. 402.

INDEX TO AUTHORS, VOL. XIV.

PAGE

Alger, Ellice M.:
Paralysis of Divergence 665

Ayer, James B.:
Lumbar Puncture and Examination of the Spinal Fluid in Affections of the Eye 205

Bedell, Arthur J.:
Report of a Case of Colloid Degeneration of the Upper Lid 313

Blake, Eugene M.:
The Etiology of Retinitis Circinata 753

Bordley, James, Jr.:
Malignant Uveitis Treated with Thyroid Extract 232
Two Patients with Unusual Corneal Opacities 527

Brown, E. V. L.:
The Etiology of Iritis 495

Bruner, William Evans:
A Case of Pituitary Body Disease with Reference to the Effects of Early Operation 632
Memoir of Dr. Benjamin L. Millikin 429

Bruns, Henry Dickson:
The Ambulant After-treatment of Cataract Extraction; with a Note on Post-operative Delirium and on Striped Keratitis 473

Capron, F. P.:
Multiple Melanotic Nevi of Conjunctiva, with Malignant Invasion of the Corneal Limbus 322

Chance, Burton:
Recurrent Sarcomata at the Limbus Treated by "Electric Desiccation" 550

Charles, J. W.:
Memoir of Dr. M. Hayward Post 15

Cheney, Frederick E.:
Memoir of Dr. Benjamin Joy Jeffries 427

Claiborne, J. Herbert:
A Review of the History of the Electric Ophthalmoscope, with a Demonstration of a New Ophthalmoscope, which Consists of a Rearrangement of the Parts of Antecedent Instruments 150

Davis, A. E.:
Spontaneous Reattachment of the Retina after Twenty-two Years' Duration; Report of Case with Remarks 376

Derby, George S.:
Massive Granuloma of the Sclera (Brawny Scleritis), with the Report of an Unusual Case 110

Duane, Alexander:
Anomalies of the Accommodation Clinically Considered 386
Indications for Tenotomy of Inferior Oblique Muscle 82

PAGE

Ewing, A. E.:
A Practical Lamp for Ophthalmic Operations 565
A Uniform Visual Test-object 566
Memoir of Dr. M. Hayward Post 15

Friedenwald, Harry:
The Ophthalmias of Palestine 264

Greenwood, Allen:
Ophthalmoscopic Work in the British Base Hospitals 529
Griscom, J. Milton:
Hereditary Posterior Polar Cataract, with Report of a Pedigree 356

Hansell, Howard F.:
A Case of Epibulbar Sarcoma 320
High Hyperopia from a Clinician's Standpoint 639
Harrower, David:
A Report of Several Cases of Chronic Simple Glaucoma Treated by Iridotasis 55
Heckel, Edward B.:
Report of a Case of Melanosarcoma of the Orbit Treated with Radium 560
Heed, Charles R.:
A Case of Primary Intradural Tumor of the Optic Nerve 331
Holloway, T. B.:
Two Cases of Orbital Growth 542
Howe, Lucien:
A More Exact Method of Measuring Fields of Vision and Arcs of Rotation 183

Irons, Ernest E.:
The Etiology of Iritis 495

Jackson, Edward:
Tuberculosis of the Retinal Vessels 577

Knapp, Arnold:
A Primary Tumor of the Optic Nerve Successfully Removed, with Preservation of the Eyeball, by the Krönlein Method 335
The Operative Treatment of Partial Staphyloma of the Cornea and of Fistula of the Cornea with a Conjunctival Flap; Conjunctivo-Keratoplasty (Kühnt) 466
Report of the Removal of a Tumor at the Apex of the Orbit with Preservation of the Eyeball in a Case of Plexiform Neuroma of the Eyelid. Microscopic Examination 534
Salvarsan in Interstitial Keratitis and in Optic Nerve Lesions 124

Lamb, Robert Scott:
Cyanosis Retinæ et Conjunctivæ in Connection with Pulmonary Stenosis and Patent Ductus Arteriosus 627
Lancaster, Walter B.:
A Case of Botulism 648
Stronger Eye Magnets 168
Langdon, H. Maxwell:
The Ives-Langdon Photometer 144
Isolated Paralysis of the Cervical Sympathetic Accompanying Goiter 661

PAGE

Marple, Wilbur B.:
Memoir of Dr. Emil Gruening 9
May, Charles H.:
The May Electric Ophthalmoscope 165

Ohly, John H.:
The Electromagnet and the Magnet Operation in Ophthalmic Surgery 762

Posey, Wm. Campbell:
Tenotomy of the Inferior Oblique Muscle 65
Pusey, Brown:
Family Degeneration of the Macula Lutea, with a Suggestion as to Its Cause 364

Randolph, Robert L.:
A Case of Blepharochalasis—A Variety of Dermatolysis 518
A Clinical and Pathological Study of Two Cases of Miliary Tubercles of the Choroid 585

Sattler, Robert:
A Review of the Glaucoma Question, with Special Reference to Newer Conclusions of Its Etiology and Departures of Its Surgical Treatment 38
Schmeisser, H. C.:
A Clinical and Pathological Study of Two Cases of Miliary Tubercles of the Choroid 585
de Schweinitz, G. E.:
Progressive Essential Atrophy of All Layers of the Iris 250
A Contribution to the Subject of Tumors of the Eyelid and Orbit ... 341
Shine, Francis W.:
The Motais Operation for Ptosis 733
Shumway, Edward A.:
A Simple Modification of the Berger Loupe 158
Snell, Albert C.:
An Epibulbar Melanosarcoma of the Limbus which Completely Disappeared under *X*-ray Treatments 556
Standish, Myles:
Memoir of Dr. Hasket Derby 1
Retrobulbar Neuritis with Central Scotoma from Toxic Action of Thyroidin 608
Stieren, Edward:
Pemphigus of the Conjunctiva.—Report of Two Cases 302
Sutphen, T. Y.:
Unusual Complications in a Case of Glaucoma after Atropin 645
Sweet, William M.:
Some Experiences in Enucleation under Local Anesthesia 98

Theobald, Samuel:
Observations upon Herpes Corneæ "Febrilis," with Reference, Especially, to Etiology 520
Tooke, Frederick:
Tuberculous Meningitis with Special Reference to the Tubercle of the Choroid and Its Pathologic Manifestations 218
Török, Ervin:
Extraction of Cataracts in the Capsule by a Slight Modification of the von Graefe Method 482

PAGE

Vail, Derrick T.:
A Discussion of Some Newer Principles in Dealing with Uncomplicated Cataract 718
Veasey, C. A.:
An Unusually Large Osteoma of the Frontal, Ethmoidal, and Sphenoidal Sinuses Involving the Orbit and Anterior Cerebral Fossa, Presenting no Subjective Symptoms Other than Proptosis of the Eyeball 538
Verhoeff, F. H.:
A Case of Syphilitic Retinochoroiditis Juxtapapillaris, with Microscopic Examination 568
Improved Capsule Forceps for Intracapsular Cataract Extractions .. 489
Pathologic Examination of a Case of Massive Granuloma of the Sclera 110
Rosacea Keratitis and Certain Other Forms of Marginal Keratitis, Neuropathic in Origin. Treatment by Pericorneal Neurotomy 89

Walker, Clifford B.:
Color Interlacing and Perimetry 684
Weeks, John E.:
Report of Cases of Quinin Amblyopia, with Remarks 593
Report of Two Cases of Metastatic Carcinoma of the Choroid and One Case of Myxosarcoma of the Orbit 326
Wheeler, John M.:
A Study of Hemorrhage into the Anterior Chamber Subsequent to Operations for Hard Cataract 742
Wilder, William H.:
Memoir of Dr. Charles Heady Beard 432
Optic Neuritis from Syphilitic Leptomeningitis 614
Williams, Charles H.:
Nagel's Anomaloscope for Testing Color-Vision 161
Williams, Edward R.:
Nervous Symptoms from Contusions of the Eye 402
Wilmer, W. H.:
Three Years' Experience in Sclerocorneal Trephining in Glaucoma .. 453
Woods, Hiram:
Ocular Phenomena Accompanying Three Cases of Gastro-Intestinal Disorder 189
Wootton, Herbert Wright:
Divergent Concomitant Strabismus, the Muscular Anomalies and Refractive Errors Accompanying It, and Its Treatment 678

Zentmayer, William:
Retinal Disease with Massive Exudation and an Allied Condition: Report of Two Cases 370
Ziegler, S. Lewis:
Hereditary Posterior Polar Cataract, with Report of a Pedigree 356
Memoir of Dr. George Strawbridge 4

INDEX TO SUBJECTS, VOL. XIV.

Accommodation, Anomalies of, 386
Amblyopia, Quinin, 593
Anomalies of Accommodation, 386
Anomaloscope, Nagel's, 161
Anterior Chamber, Hemorrhage into, 742
Arcs of Rotation, Exact Method of Measuring, 183
Atropin, Glaucoma after, 645

Beard, Charles Heady, Memoir of, 432
Berger Loupe, Modification of, 158
Blepharochalasis, Case of, 518
Borthen Operation for Glaucoma, 55
Botulism, 648
Brawny Scleritis, 110
British Base Hospitals, Ophthalmoscopic Work in, 529

Carcinoma, Metastatic, of Choroid, 326
Cataract, Ambulant, After-treatment of, 473
Extraction in Capsule, 482
Hereditary Posterior Polar, 356
Newer Principles in Dealing with, 718
Cerebral Fossa, Osteoma of, 538
Cervical Sympathetic, Paralysis of, 661
Choroid, Metastatic Carcinoma of, 326
Miliary Tubercle of, 218, 585
Colloid Degeneration of Upper Lid, 313
Color Interlacing and Perimetry, 684
Color-vision, Nagel's Anomaloscope for Testing, 161
Conjunctiva, Cyanosis of, 627
Melanotic Nevi of, 322
Pemphigus of, 302
Conjunctival Flap for Fistula of Cornea, 466
Conjunctivo-keratoplasty, 466
Contusions of Eye, Nervous Symptoms from, 402
Cornea, Fistula of, 466
Herpes of, 520
Staphyloma of, 466
Unusual Opacities of, 466
Cyanosis Retinæ and Conjunctivæ, 627

Degeneration of Macula Lutea, 365
Delirium, Post-operative, 473
Derby, Hasket, Memoir of, 1
Dermatolysis, 518
Detachment of Retina, Spontaneous Reattachment of, 376
Divergence, Paralysis of, 665
Divergent Strabismus, 678

Electric Desiccation, 550
Electric Ophthalmoscope, History of, 150
Electromagnet, 762
Enucleation under Local Anesthesia, 98
Epibulbar Lipoma, 353
Sarcoma, 320, 556
Ethmoidal Sinus, Osteoma of, 538
Extraction of Cataract in Capsule, 482, 489
Eyelid, Colloid Degeneration of, 313
Myxosarcoma of, 341
Plexiform Neuroma of, 534
Tumors of, 341
Eye Magnets, Stronger, 168

Fields of Vision, Exact Method of Measuring, 183
Fistula of Cornea, 466
Frontal Sinus, Osteoma of, 538

Gastro-intestinal Stasis, Ocular Phenomena of, 189

Glaucoma After Atropin, 645
Etiology and Surgical Treatment, 38
Iridotasis Operation, 55
Sclerocorneal Trephining in, 453
Goiter, Paralysis of Sympathetic Accompanying, 661
Granuloma of Sclera, 110
Gruening, Emil, Memoir of, 9

Hemorrhage into the Anterior Chamber, 742
Hereditary Posterior Polar Cataract, 356
Herpes Corneæ "Febrilis," 520
Hyperopia, High, 639

Inferior Oblique Muscle, Tenotomy of, 65
Interstitial Keratitis, Salvarsan in, 124
Intracapsular Cataract Extraction, 482, 489
Intradural Tumor of Optic Nerve, 331, 335, 347
Iridotasis for Glaucoma, 55
Iris, Progressive Atrophy of, 250
Iritis, Etiology of, 495
Ives-Langdon Photometer, 144

Jeffries, Benjamin Joy, Memoir of, 427

Kalt Capsule Forceps Modified, 489
Keratitis, Interstitial, Salvarsan in, 124
Marginal, 89
Striped, 473
Keratoplasty, Conjunctivo-, 466
Krönlein Operation, 335, 534

Lacrimal Gland, Tumor of, 542
Lamp, Ophthalmic Operating, 565
Leptomeningitis with Optic Neuritis, 614
Limbus, Recurrent Sarcoma of, 550
Sarcoma of, 556
Lipoma, Epibulbar, 353
List of Members, v, 421
Local Anesthesia in Enucleation, 98
Loupe, Modified Berger, 158
Lumbar Puncture in Eye Affections, 205

Macula Lutea, Family Degeneration of, 364
Magnet Operation, 762
Magnets, Eye, Stronger, 168
Massive Exudation of Retina, 370
Granuloma of Sclera, 110
Meningitis, Tuberculous, 218
Metastatic Carcinoma of Choroid, 326
Miliary Tubercle of Choroid, 218, 585
Millikin, Benjamin L., Memoir of, 429
Minutes of the Proceedings, 21, 437
Motais Operation for Ptosis, 733
Myxosarcoma of Eyelid, 341
of Orbit, 326

Nagel's Anomaloscope for Testing Color-vision, 161
Nervous Symptoms from Contusions of Eye, 402
Neuritis, Retrobulbar, from Thyroidin, 608
Neuroma of Eyelid, 534
Neurotomy, Pericorneal, 89
Nevi of Conjunctiva, 322

Operating Lamp, 565
Ophthalmias of Palestine, 264
Ophthalmic Lamp, 565
Ophthalmoscope, Electric, 150, 165
Optic Nerve, Lesions, Salvarsan in, 124
Primary Intradural Tumor of, 331, 335, 347
Psammo-endothelioma of, 347
Optic Neuritis, Syphilitic, 614
Orbit, Melanosarcoma of, 560
Myxosarcoma of, 326
Osteoma of, 538
Sarcoma of, 344
Tumors of, 341, 534
Osteoma of Sinuses, 538

Palestine, Ophthalmias of, 264
Paralysis of Cervical Sympathetic, 661
of Divergence, 665
Pemphigus of Conjunctiva, 302
Pericorneal Neurotomy, 89
Perimetry, 684
Photometer, Ives-Langdon, 144
Pituitary Body Disease, 632
Plexiform Neuroma of Eyelid, 534

Post, M. Hayward, Memoir of, 15
Post-operative Delirium, 473
Progressive Atrophy of Iris, 250
Psammo-endothelioma of Optic Nerve, 347
Ptosis, Motais Operation, 733

Quinin Amblyopia, 593

Radium Treatment of Sarcoma of Orbit, 560
Retina, Cyanosis of, 627
Massive Exudation of, 370
Spontaneous Reattachment of, 376
Tuberculosis of Vessels of, 577
Retinal Detachment, Spontaneous Reattachment of, 376
Retinitis Circinata, Etiology of, 753
Retinochoroiditis Juxtapapillaris, Syphilitic, 568
Retrobulbar Neuritis, 608
Roentgen Ray Treatment of Sarcoma, 556
Rosacea Keratitis, 89

Salvarsan in Interstitial Keratitis, 124
in Optic Nerve Lesions, 124
Sarcoma, Epibulbar, 320
of Limbus, 550, 556
of Orbit, 344, 542, 560
Sclera, Massive Granuloma of, 110
Scleritis, Brawny, 110
Sclerocorneal Trephining in Glaucoma, 453
Sinuses, Osteoma of, 538
Sphenoidal Sinus, Osteoma of, 538
Spinal Fluid, Examination of, 205
Staphyloma of Cornea, 466
Strabismus, Divergent, 678
Strawbridge, George, Memoir of, 4
Striped Keratitis, 473
Syphilitic Leptomeningitis, 614
Retinochoroiditis Juxtapapillaris, 568

Tenotomy of Inferior Oblique Muscle, 65
Test-object, Uniform Visual, 566
Thyroid Extract for Malignant Uveitis, 232
Thyroidin, Retrobulbar Neuritis from, 608
Trachoma in Palestine, 264
Trephining, Sclerocorneal, in Glaucoma, 453
Tubercle, Miliary, of Choroid, 218, 585
Tuberculosis of Retinal Vessels, 577
Tuberculous Meningitis, 218
Tumor of Orbit, 534
of Pituitary Body, 632
Tumors of Orbit, 341

Uveitis Treated with Thyroid Extract, 232

Visual Test-object, 566

RE
1
A55
v.14

American Ophthalmological Society
Transactions

Biological
& Medical
Serials

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY

STORAGE